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# Computational Study Of Fluid-Valve Interaction Involving Deep Vein Thrombosis

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INVOLVING DEEP VEIN THROMBOSIS

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# Abstract

The venous valves in veins carry on the important function of eliminating the retrograde (reverse) flow of blood in veins and help the veins to carry on their work effectively. The abnormal functioning of venous valves can cause deep vein thrombosis (DVT) which is a phenomena caused by clotting in deep veins. The importance of the thesis work lies in understanding the role of blood flow dynamics in Deep Vein Thrombosis (DVT). Elderly People and pregnant women are the ones who are most likely to develop DVT. It was determined from previous studies that women in their postpartum period are at the highest risk of developing a DVT and even pulmonary embolism in most of the cases. The aid of computational study could help us in improving the prophylaxis given to the elderly and pregnant women, by better understanding the physics involved in the clot formation.

The main goal of my thesis work is to study fluid dynamics behavior in deep veins with computational fluid dynamics. For this work, I create a geometry which resembles the blood vein containing unidirectional venous valves and also I am going to create the same flow circumstances which depict the flow undergoing in the blood veins. With latest advancement in computational resources (both software and hardware), it has become affordable to perform high fidelity computational studies for this problem.

The main point of interest is to see what happens to the flow patterns when the blood flows past the valves in the blood veins and to understand how the velocity varies in the pocket like structure. My thesis work lets us see the flow patterns in the blood veins and find where the vortices are formed when there are obstructions like the valves acting to stop the flow, which could be helpful in determining a chance for the clot formation in some specific areas.

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# Chapter1: Introduction

Every year in the United States complications from Deep Venous Thrombosis (DVT) results in 250,000 hospitalizations and over 50,000 deaths. The general risk of venous thromboembolism (VTE) (one disease with manifestations of both pulmonary embolism (PE) and DVT) in general medical patient ranges from 10 to 30% (Meyer-Michel 2000) (Fredrick and Fredrick 2003). It is the third most prevalent cardiovascular disease and remains the most preventable cause of death in our hospitals. Also, reports suggest that the number of hospital admissions in 1990's ranged from 300,000 to 600,000 and the death toll was approximately 50,000 per year (Herbert Janssen 1993).

The risk of DVT and its associated conditions such as PE and chronic venous insufficiency (CVI) increase with age (Stein, et al. 2004). It has been demonstrated in previous studies that venous valves in the legs of elderly individuals are often damaged or missing. This damage occurs as a leaflet tears, an ancillary flaps, a leaflet develops or grows into the vein wall, and/or as remnants of the leaflet that appear as only small outgrowths on the vein wall.

These abnormalities generally result in CVI since valve incompetence most frequently exists in these conditions. The damage also inhibits proper valves function, which enhances the risk of blood stasis; one of the major factors that increase the risk of future DVT development. The flow in the deep veins takes place with the help of the muscles and the vein valves.

The muscles squeeze the veins and help the blood to flow against the gravity and vein valves help in restricting the flow from flowing in the opposite direction. When the muscles contract, they pressurize the veins which intern squeezes the blood to flow against the gravity and when this happens the venous valves move away from each other allowing the blood to flow through the valves. When the muscles relax the blood tends to flow back due to gravity so the venous valves come closer to each other and form a seal. Venous valves eliminate the retrograde flow of the blood; hence they are called unidirectional valves.

As blood flows through the arteries and veins, a balance is continuously maintained between factors that induce clots (procoagulants such as fibrin, fibrinogen, prothrombin, etc.) and other factors that prevent clots from forming (anticoagulatants such as antithrombin-III, activated protein C, fibrinolytic enzymes, etc). In such a system an increase in activity (e.g. an increase clotting rate) can be achieved by either increasing the procoagulants or decreasing the anticoagulants. At first glance this seems wasteful; however, in the human body, this assures that the clot forming system is always prepared to respond quickly to an injury that would otherwise produce a life threatening blood loss (Bertina 2009) (Paul and Sabine 2005).

Elderly People and the pregnant women are the ones who are most likely to develop a DVT. From previous studies (Heit, et al. 2005) it was determined that women in their postpartum period are at the highest risk of developing a DVT and even pulmonary embolism in most of the cases. This is because; during pregnancy and postpartum there is an increased level of coagulation or clotting factors in the blood. Also there is an increased venous diameter which intern reduces the flow of blood from the lower extremities (Wee-shian and Jeffrey 2002). Apart from elderly people and the pregnant women, stroke patients are the ones who are at a higher risk of developing a DVT (Georgia, Trevor and Philip 1995).

The aid of computational study could help in improving the prophylaxis provided to all the people at risk of DVT, especially those whose increased risk is related to venous stasis, by better understanding the physics involved in the clot formation. The modeling will prove to be a valuable resource to better understand this observed condition that has continued to threaten the lives of our elderly. This study would help us know how the flow develops in the deep veins and also how it varies with the varying distance of the valves that is the opening and the closing of the valves. We could get a detailed understanding of where the vortices would be formed for different valve positions.

The second chapter discusses the literature which explains in detail about the arrangement, functionality and other such properties of the veins, shows what are the different phenomena that occur in deep veins and how they can be handled and in the later part of this chapter shows how computational analysis is being used as an important analysis tool in the field of medicine.

The third chapter discusses in detail the methods used in creating the geometry, the equations solved by the solver, the kind of solvers used, the type of grids created and also how the problem is divided into several cases and solved. It also gives some information about the kind of results being captured.

The fourth chapter discusses the various results of the number of cases into which the problem was divided and also shows the velocity and pressure profiles of each of these cases and explains about these contours in detail.

The fifth and sixth chapters discuss the conclusions made and then discuss scope of future work that can be done in this field of study respectively.

# Chapter 2: Literature Survey

This chapter mainly focuses on giving a broader picture of research that was previously done to show how veins function, how the valves work, what are the diseases that could occur in veins and valves and it also explains how the aid of computational tools has helped in bettering the medical industry.

## 2.1 Veins, Venous valves and Deep vein Thrombosis (DVT):

Blood vessels differ in their architectural structure and they behave differently to handle different tasks (Bader n.d.). The blood vessels which carry the oxygenated blood from the heart to different parts of the body are called arteries and the vessels which carry de-oxygenated blood from body parts to the heart are called veins.

Veins are an integral part of the human body. Also the functioning of the veins is much more complex when compared to the function of the arteries. Veins in the leg can be considered as long thin collapsible tubes containing a series of one-way bicuspid valves (Buescher, et al. 2005). The veins carry on the important function of returning the deoxygenated blood from all the parts of the body back to heart (right side of the heart) (Anne July 2001). There are two major types of veins: the superficial veins and the deep veins. As the name suggests the superficial veins are the ones which are just under the layer of skin and are visible in green color and the deep veins are the one's which are deep inside the muscle tissue.

There are a third kind of veins called the perforating veins. As their name suggests, they are present in between the superficial veins and the deep veins. The main function of these veins is to connect the superficial veins with the deep veins and to ensure that the flow remains unidirectional within the veins: from the superficial veins to the deep veins. These kind of veins are further classified as direct perforators and the indirect perforators, the direct perforators connects the superficial and the deep veins while the indirect perforators connect the other veins in the muscles to the superficial veins in order to effectively draw blood from the superficial veins.

The deep veins are present in the lower body and they help drain the blood from the lower extremities. The deep veins of the leg are covered well by the muscle and are supported by the tissue surrounding them; these veins produce 90 to 95% of the venous outflow from the leg (Anne July 2001).

Effective venous return from the lower extremities requires the interaction of the heart, a pressure gradient, the peripheral muscle pumps of the leg, and competent venous valves (Mark, et al. 2007). There are several unidirectional valves in the deep veins which function in coordination with the muscles in order to eliminate any retrograde flow of the blood and help the veins to function effectively when pumping the blood against gravity.

*Venous Valves:* The venous valves are one of the smallest and the most delicate organs present in the human body. They are present in the blood veins and are bicuspid in nature i.e. they have two flaps. When they where first discovered, it was presumed that their function was to form an obstacle to the blood flow.

Later it was found that venous valves carry on the important function of eliminating the retrograde (reverse) flow of the blood in the veins and help the veins to carry on their work effectively. From then onwards, the venous valves where considered important in the concept of the circulatory system. Each valve has a couple of very thin walled cusps which originate at the opposite sides of the wall (vein wall) and meet at the center. They are able to regulate the flow and keep it unidirectional when the pressure above the valves is greater than the pressure below them.

When the muscles contract, these venous valves move apart. There by allowing flow of blood towards the heart. When the muscles expand, these valves move close to each other blocking any reverse flow of the blood in the veins. Hence, the greater the pressure of the circulating fluid, the greater will be the opening of the valves. Thus these venous valves are able to eliminate any retrograde flow of blood in the blood veins unless there are any disturbances within veins like the clots (Franklin 1927).

The veins of less than 1 mm diameter do not contain these valves. The distance between the valves in the deep veins is much shorter when compared to that in the superficial veins. Also, the alignment of valves in perforating veins helps ensure the unidirectional flow of blood from the superficial veins to the deep veins.

Figure 1: Arrangement of venous valves (Gottlob and May n.d.)

The valves are of two types namely parietal valves and the ostial valves. The valves which are most frequently encountered are the parietal valves. These parietal valves are otherwise called the pocket valves and contain cusps. The ostial valves occur less frequently and are placed at the intersection of small veins and large veins especially at the places where the small veins transform into the large veins. The parietal valves consist of cusps, the valvular agger, the valvular cornua and the valvular sinus. The valvular cusps intersect at the valvular agger and are usually half moon sharpened like structures in shape (Franklin 1927).

The venous valves that are found in the veins have cross sections of elliptical outline at the sites of the venous valves. The formation of these valves takes place very early in the life cycle, in fact the earliest formation of the valves was seen in the embryos that were just 3.5 months old. In a 70 years old adult only 19 to 20 percent of the total valves exist. This reduction in the number of venous valves can be due to thromboses of valvular sinus which occurs more frequently than it actually is taught to occur. Further studies show that these valves just do not disappear but, they become impotent along with the increasing age. This impotence of the venous valves might be due to their over dilation of venous valves as well as massive venous thrombosis or even due to over-dilation of venous wall.

Some experiments were carried out to see how these valves functioned, in one of the experiment a probe was attempted to be passed through the connecting veins to see whether the valve functioning can be studied. The experiment showed that when the probe was passed from the smaller branches of veins to the deep veins it would not be possible to move the probe, and the deep veins always resisted the probe. But when the probe was inserted in the reverse direction i.e. from the deeper veins to the branches, the veins did not offer any resistance. This study showed that the blood flow is regulated in a single direction from superficial veins to deep veins.

Factors like high pressure on valves especially in the persons whose jobs require long time standing, their veins are compressed for long periods of time by the compressed muscles. Also, during pregnancy increased dilatability of vessels was observed. One incompetent valve could lead to the overloading of the other valves i.e. the valves might have to handle increased amount of blood flow, such cases can lead to the dilation of the walls.

When the walls of the veins get dilated the retrograde flow of the veins cannot be stopped. This in turn leads to the over-dilation of the walls which makes the venous valve incompetent (Robert 1980). The over-dilated walls and incompetent valves lead to blood regurgitation and turbulences. These turbulences could cause further complications which render the valve incompetent (Gottlob and May n.d.).

*Deep Vein Thrombosis (DVT):* Usually, clots are formed at the places where there are any cuts or wounds. These occur in order to prevent excessive loss of blood which essentially makes them useful clots and without such clots life would be difficult. Thrombus means a clot and the formation of these clots in the deep veins is called the deep vein thrombosis. There is a possibility that these clots block blood from flowing to the heart. Sometimes these clots prevent the one way venous valves from closing this in turn makes the blood flow in the reverse direction. This further leads to the swelling of the legs or the effected parts of the body. As DVT occurs mostly in legs, the elderly and pregnant women are the ones who are most prone to developing it (Heit, et al. 2005).

It is a well accepted that DVT forms in the pockets located behind the venous valve leaflets. These thrombi which are formed in the pockets, progress into larger clots that can break loose and form Pulmonary Emboli (PE). It is also known that the clots in these pockets can damage the leaflets resulting in incompetent valves incapable of closing properly. Increased venous pressure, peripheral edema, and ultimately venous ulcers will result if the condition is allowed to progress. These ulcers are not life threatening, but they significantly reduce the quality of an individual's life.

*Clot formation:* The process in which the blood transforms into a clot or thrombi is called a coagulation or hemostasis. When a wound is formed and bleeding starts, a substance called fibrin is released by the action of two clotting factors in the blood called procoagulants and anticoagulants which prevents further bleeding by clotting the blood. This process of clotting of the blood is called hemostasis (Bertina 2009).

Hemostasis of blood occurs in three stages: an initiation phase, a propagation phase and a termination phase. During the initiation phase, the concentration of thrombin tends to increase slowly. During the propagation phase, thrombin activates platelets which in turn support the procoagulants reactions. It is this propagation phase which plays a major role in the regulation of the clot formation. In the termination phase, the hemostatic process (i.e. the formation of clot) occurs.

*Clots could form due to:*

Inactivity of certain body parts (especially legs), which are absolutely motionless during a long flight journey, could be a potential cause for developing a DVT. This is because when the legs are motionless the leg muscles have to contract more in order to pump the blood from the legs to the heart and the valves have to prevent the retrograde flow of the blood. This takes more effort when the legs are absolutely motionless.

Hypercoagulation and surgeries are a couple of other factors which play a major role in the clot formation. In the case of hyper coagulation, the blood has the tendency to clot more than it is normally expected to. In the case of surgeries, the drugs given to the patient could sometimes alter the clotting factors of the blood. Again, hyper coagulation might occur either hereditarily or by the use of drugs.

*Virchow's Triad*:

Rudolph Virchow recognized a triad which is immensely helpful in understanding the clotting system of the human body (Joseph, Edward and Robert 1948). The three factors that contribute to the development of the clot according to the triad of Virchow are (Anthonie, et al. 1999):

- Venous stasis
- Clotting Factors
- Vessel damage

According to Virchow's Triad there should be a presence of at least two of the three factors in the triad in order for a clot to develop. Virchow's Triad has laid a path for the clinicians to develop methods that would reduce the clotting of blood (N. S. Wijeratne 2008).

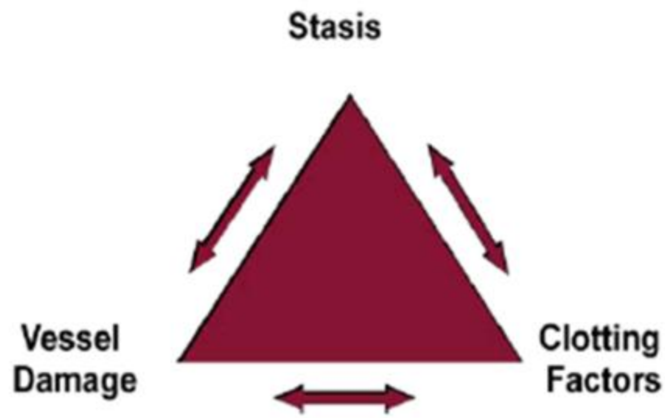


Figure 2 : Virchow's Triad

**2.2 Complications which might occur apart from a DVT:**

*Pulmonary Embolism:* This is one of the more serious conditions that could occur due to the clots formed in the veins. This occurs when the clot in the blood veins happens to travel along with the blood stream into the heart and then into the lungs. After the clot reaches the lungs it gets deposited in a region of the lungs and makes that particular region of the lung ineffective by blocking the pulmonary artery. Pulmonary embolism is regarded as one of the most common causes of death after a surgery (Kakkar, et al. 1970).

Pulmonary embolism is serious enough to cause death in some of the cases. It causes death in over 100,000 patients each year in the United States and is also a main contributing factor in another 100,000. With this being said, PE might be the most preventable cause of death (especially after surgery) in hospitals (Clagett, Anderson Jr, et al., Prevention of venous Thromboembolism 1992) (Jerry and Wolfgang 2004).Some studies even concluded that the development of DVT and PE are very much interlinked. This was proven by an experiment which resulted in reduced PE with the decrease in the number of DVT (Janseen, et al. 1986).

Chronic Venous Insufficiency: Sometimes the clots formed in the venous valves become so strong that they can easily cause damage to the venous valves rendering them impotent. The damaged venous valves are unable to close properly; as a result, they are unable to carry on their function of eliminating the retrograde flow of the blood effectively: called Chronic Venous Insufficiency (CVI). The prevalence of CVI ranges from 2 to 7% in men and 3 to 7% in women (Abbade and Lastoria 2005).

*Venous Ulcers:* A venous ulcer is generally an ulcer (wound) which is irregularly shaped and which initially develops superficially, but has the potential to deepen, with well defined borders. These are usually caused due to an ill functioning venous valve, which can arise due to CVI. The venous ulcers are usually very difficult to heal and it is also a common problem in the adult population. The prevalence of venous ulcers ranges from 40 to 50% in males and from 50 to 55% in females (Abbade and Lastoria 2005).

*Methods of diagnosing a DVT:*

Both DVT and PE show few specific symptoms and clinical diagnosis is insensitive and unreliable (Clagett, Anderson Jr, et al., Prevention of venous Thromboembolism 1992).The methods of diagnosing a DVT are of two major types called the invasive techniques and non invasive techniques. In the invasive techniques, an incision is made in order to get to the affected regions. This induces pain and other side effects while the non invasive technique requires no such process for the diagnosis because most of the noninvasive techniques involve imaging the affected area. A study stated that the help of non-invasive diagnostic tests have improved the diagnostic procedures of detecting a suspected DVT in patients (Philips, et al. 1995). A few of the diagnosis methods are discussed below (Eran and Edwin 1994),

*Physical Examination:* A physical examination is done on the legs; also the blood pressure is checked. This kind of test is not very effective for detecting a DVT but can be used as a screening test in order to determine if any further action is required.

*Venography*: In this test a dye is introduced into the deep vein, and the x-ray of the vein in which the dye is introduced is taken. The main reason for introducing the dye into the vein is that it makes that particular vein visible under the x-ray. Lower extremity contrast Venography was regarded as a gold standard for the diagnosis of DVT in early 90's (M.M.W, Van and Ten 1994).

*Ultrasound examination*: In this, a transducer is used to detect the clot in leg veins. The sound waves are passed through the veins where the clot is expected and outputs are analyzed to determine whether the clot exists.

*D dimer blood test*: This test is used to determine clotting factors of the blood. This is helpful for understanding the risks of an individual to be affected with a DVT.

*CT and MRI Scanning*: Both of these scanning reports provide pictures which will be helpful for detecting the clot.

*Venous Occlusion Plethysmography* (VOP) is a noninvasive technique widely used for the detection of deep vein thrombosis. The presence of major venous outflow obstruction is determined by comparing the rate of venous outflow with the maximum calf volume change.

Apart from these techniques there are several other techniques in the present day to test for a DVT.

Studies on pregnant women and their relative risk of venous thromboembolism (Heit, et al. 2005): A population based study in Olmsted County, Minnesota was conducted to study the incidence of venous thromboembolism in pregnant women. The study showed that, 105 cases of DVT or PE were identified in women who were either pregnant or women who were in the postpartum period.

These figures were said to be 4 times higher than the expected number of DVT and PEs found in women who were not pregnant. It was also determined that the overall incidence of venous thromboembolism was higher in postpartum period than during the pregnancy. Also the same study states that the incidence of venous thromboembolism in the general population is strongly and directly related to the increasing patient age.

*Factors responsible for causing a venous disease:*

In 1986, The National Institutes of Health (NIH) held a Consensus Development Conference entitled: Prevention of Venous Thrombosis and Pulmonary Embolism (National 1986). Despite being conducted almost 25 years ago, the proceedings of the conference remain as the foundation for our understanding of thromboembolic disease. Since that time others have added to the literature, but the original contribution of the NIH conference is considered a pivotal point in our effort to better understand and treat DVT and PE. Several of the most important contributions made by the conference include: 1) defining the extent of the problem, 2) identifying the risk factors of thromboembolic disease, 3) reinforcing the link between DVT and PE, 4) and identifying types of prophylaxis that would be most suitable for each group. Many of the recommendations made by the consensus development continue to be used by researchers and clinicians alike.

Shortly after the NIH Consensus Development Conference, a computerized epidemiological approach was developed to help predict the risk of DVT (Herbert, et al. 1986). This program relayed heavily on the NIH Consensus Development Conference report to identify the risk factors. It added information taken from previous literature to assign a numerical risk to each condition when possible. One such reference was an epidemiological work completed by W. W. Coon some years earlier (Coon 1977). The risk factors included in the computer model included:

1. Previous history of deep venous thrombosis, 2. Varicose veins, 3. Increasing age, 4.Obesity, 5. Venous stasis, 6. Heart disease, 7. Cancer, 8. Surgical procedures, 9. Pregnancy and the post-partum period, 10. Oral contraceptives, 11. Trauma, 12. Chronic ulcerative colitis, 13. Infections, 14. Myeloproliferative disorders, 15. Lupus anticoagulant, 16. Nephritic syndrome, 17. Paroxysmal nocturnal hemoglobinuria, 18. Antithrombin III deficiency, 19. Protein S and protein C deficiency and 20. Abnormal plasminogen activity. Some of these risk factors produce little effect; however, others such as cancer, major surgery, advancing age, pregnancy, and a previous history of DVT appear to significantly increase an individual's future risk. Individuals with these risk factors should remain alert to the possibility of thromboembolic disease and should seek medical attention to prevent this detrimental condition.

Since the time of this article other factors have also been identified. The most notable was the identification of Leiden factor V as an inherited cause of DVT risk. The risk is found in approximately 5% of the Caucasian population and contributes to an additional increased risk of clotting previously identified risk factors such as pregnancy, surgery, stasis, cancer, and oral contraceptive use. As with many other factors that increase the risk of DVT and PE, the contribution of Leiden V to other factors may be less that the combined sum, additive, or synergistic (Donahue 2004).

**2.3 Computational Studies:**

Use of CFD as a tool to study the flow dynamics of the blood has been taking place recently; several studies on blood flow in arteries (David 2002), as well as analysis of lungs, etc. All these studies help in improving the prophylaxis given to the patient by explaining the flow physics in detail with the help of the simulations. Also, in some cases, patient specific models are being used to carefully analyze the physics involved. It can be said that CFD can be used as a useful noninvasive technique to diagnose the patients of DVT in the future.

One of the studies built a coupled momentum method for the blood flow in the arteries. In this study, the deformation of the artery along with the flow of the blood was studied. The model was created such that the flow of blood at each stroke of the heart came into the arteries at different flow pressures and different flow velocities. The flow inside the arteries for each stroke was studied along with the movement of the artery due to that flow (Alberto, et al. 2006).

The flow dynamics are important in the treatment of the patients with aneurysms (Nurullah, et al. 2005). These aneurysms develop due to the weakening of the valves and usually form balloon like bulges in the arteries. With the help of computational analysis, the blood flow dynamics of these aneurysms can be better understood. A study used computational analysis as a tool for the treatment of cerebral aneurysms. The study used volumetric image data from which they created the lumen geometry. Also, the boundary conditions for the finite element CFD model where taken from the same data.

The analysis thus revealed that vortices were formed due to high velocities at the proximal and distal ends; thus producing elevated wall shear stresses along the sidewalls of the aneurysm. This study concluded that image based CFD analysis can be used to provide key information about the hemodynamics for future studies on aneurysm growth (David, Jaques, et al. 2003).

Computational analysis was also done on a human carotid arterial bifurcation. In this study, the pulsatile flow in human carotid bifurcation was simulated numerically (Juan, et al. 2002). They used non invasive techniques to determine the pressure and mass flow waveforms in these artery bifurcations. The mesh and the geometry of the model were created from the MRI angiograms of the carotid artery. The flow in the carotid arteries was induced primarily by the asymmetry and the curvature of the geometry, thus they carried out the flow simulations by assuming the walls to be rigid. One of the main assumptions that were made in this study was that fluid blood was considered a Newtonian fluid. The results of the analysis showed that, the magnitude of the wall shear stress varied with the location and the cardiac cycle. It also showed that there were regions of low wall shear stress (Zaho, et al. 2000).

The main reasons for using the computational analysis over other conventional techniques are - computational analysis facilitates a detailed understanding of the flow physics involved by solving the flow equations computationally. Also, the conditions can be changed and the results can be analyzed using the computational softwares, which cannot be done physically because it is difficult to predict lot of parameters physically.

If there are any changes to be done in a system, computational analysis can exactly show whether the changes done would be beneficial to the system or not. In other words, computational analysis could make the prophylaxis given to the patients be more efficient and even safer.

## 2.4 Blood Flow and Fluid Mechanics

Fluids are broadly classified into two main types, namely Newtonian Fluids and Non-Newtonian Fluids. The Newtonian Fluids are the fluids which exhibit a linear relation between stress and strain, hence the dynamic viscosity coefficient of a Newtonian fluid is independent of time or strain measured from any reference state. The fluids in which the relation between the stress and strain is complex are called Non-Newtonian Fluids (James 2006) (N. S. Wijeratne 2008). Blood is considered to be a Non-Newtonian fluid.

The fluid flow through pipes or tubes is of interest due to its similarity to that of fluid flow in veins, arteries, bronchial air ways, vocal cords, etc. Fluid flow is classified into two types based on its behavior. The two types of flows are laminar flow and turbulent flow. The word laminar comes from the movement of adjacent fluid particles together in “laminae” (N. S. Wijeratne 2008) (John and Yunus 2008).

Laminar flow, otherwise called a streamline flow, is the flow in which the particles of fluid flow from a zero velocity at the walls to the maximum velocity in the axial stream (Helps and McDonald 1954). The particles in the laminar flow move in a well defined path and are predictable. In turbulent flows, the particles of the fluid follow a random path. One can observe the mixing of the fluid particles, especially in cases with higher velocities. A flow that alternates in between laminar and turbulent flows is called transitional flow.

The turbulences or the vertices in the blood flow are said to play a very decisive role in the development of clots. Some turbulence is found usually at the junction of smaller veins and larger veins or even at the junction of veins of equal diameters.

When there is an obstacle in the fluid flow – three eddies, one in the front and two behind the obstacle will be formed. Also a collapsed behavior of the flow is observed when an external pressure is applied to the veins.

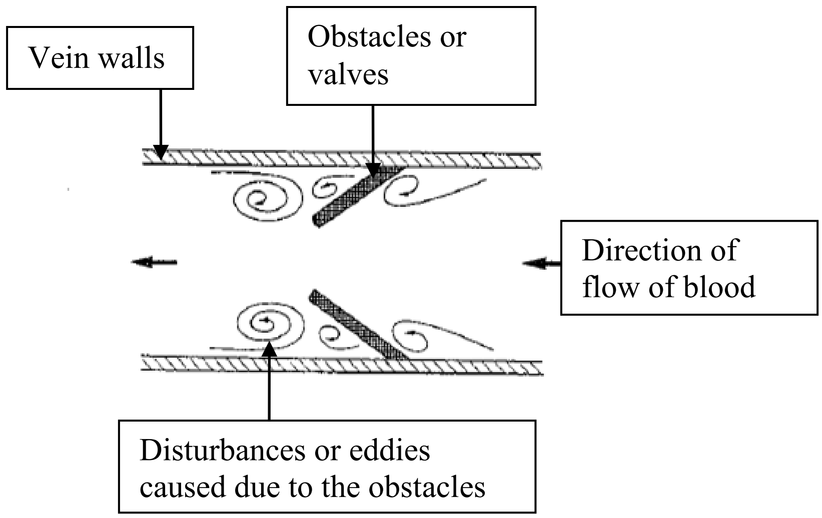


Figure 3: Flow Physics explaining the fluid flow in the presence of obstacles (Gottlob and May n.d.)

There is lot of similarity between fluid flow through the collapsible channels and the flow of fluid (blood) in the veins. The collapsed behavior can be seen in the flow in veins when an external pressure is applied to the fluid flow in veins (Wijeratne and Hoo 2006). Total irreversible collapse of the system may occur if there is any malfunctioning in the valves, or there are clots (thrombi) in the vessels. The knowledge of the flow physics involved in the venous valve could provide a key to an improved clinical treatment and to the future of designing and developing artificial valves (Yuchen, et al. 1995).

# Chapter 3: Methodology

This Chapter discusses in detail about the steps involved in modeling the problem and also about the various equations which the softwares solve for developing the flow in the model. It also focuses in detail about the meshes created, solvers used, boundary conditions applied and the results being captured.

### 3.1 Measurements of Deep Veins:


Figure 4: A scale kept by the side of a vein (Courtesy Dr. Herb Janssen)

To get the dimensions of a human vein, the vein was cut longitudinally at the junction where the two leaflets intersect at the vein wall and was kept beside a scale to get the measurements. These measurements where used to approximate the distances at each points and also the distances between the vein valves when the valves where at different positions. The dimensions which were thus measured and approximated were incorporated in the model.

Based on the above diagram the dimensions of the veins are estimated and the model is created. The co-ordinates are created, which are then connected with the help of lines and curves wherever required. A 2-d model was first created which resembles the above picture in dimensions. The position of the valve was estimated and different models were created for each position of the valves right from the valves at the fully open position to the valves almost trying to close.

Each of the case (different position of the distance between the valves) is meshed based on the complexity of the geometry. A boundary layer is added at the valves in order to capture the flow physics at the valves more effectively. Similarly a finer mesh is obtained at the areas in between the valves and a coarser mesh is made at the entrance and the exit region. An extension channel was added at the top and the bottom. The reason for doing this at the bottom was to ensure that the flow is fully developed before it enters into the model and at the top was to eradicate any possible reverse flows and ensure that the flow flows out of the system.

**3.2 Problem Definition:**

We are going to model the deep veins in which the valves are at different positions and study the flow physics of these models in order to relate this to the real life problem. Computationally modeling the flow physics involved in these deep veins helps us understand in more detail about the areas which are more susceptible to clot formation. Most of the diagnostic methods show the clots formed, but if we are able to build all the conditions perfectly we can predict the chance of clot formation more effectively and also there is always a chance to build some patient specific models which helps the clinicians decide whether there is a chance of clot formation or if there is a chance that an already formed clot can develop.

It can also play a crucial role in helping the clinicians decide whether the patient needs a surgery due to the growing clot or the clot can potentially be of no harm. Also use of CFD in most of the cases is cost effective because there is no need to use complicated machinery and also it does not need literally any maintenance costs.

So our effort is to computationally design the deep veins and analyze the flow physics in these models by applying the boundary conditions which exactly depict the flow conditions in the deep veins. We are creating the models in both 2D and 3D. To solve this problem we are using NX-Unigraphics for designing the veins in 3D and gambit to design in 2D. To build the gird we are using gambit for both 2D and 3D. Then we use fluent to computationally solve for the flow equations and finally we post process and analyze the results again by using fluent.

The figure below shows the figure obtained by creating the geometry using the co-ordinates, lines and curves in 2D. A line (y=1.2) is also shown in the figure which will be used in the later chapters in order to plot some useful graphs at different points on the line.

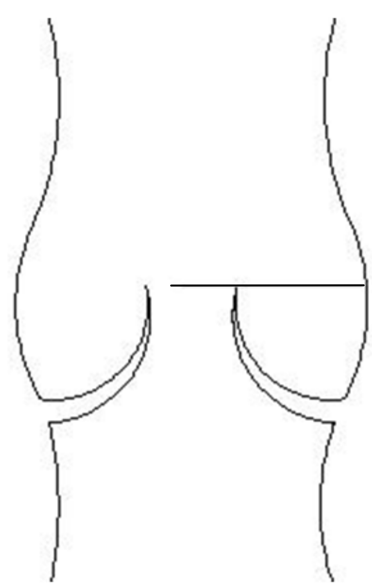

Figure 5 : Picture showing the geometry created from the above data

### 3.3 Boundary Conditions:

The gambit preprocessor software is the tool used to create the geometry and also to create the mesh (grid) for each case. The lower boundary or the line from which the fluid is desired to be entering the model is determined to be the velocity inlet of the model. The top boundary or the top line is the boundary from which the fluid leaves the model; hence it is determined to be the outlet. All the remaining regions of the model are assigned to be walls.

The model thus created has assigned velocity inlet, walls and the pressure outlet using the gambit preprocessor. However the values of the velocities and pressure at the boundaries are assigned using the fluent solver.

The fluid is considered to be entering from the bottom boundary with a velocity of 0.25m/s. Hence the bottom boundary is given an inlet velocity of 0.25 m/s in the Y-direction.

All the interiors are not having any fluid velocity, hence the interior parts, i.e. the walls and the valves of the veins are considered as walls or in other words a no slip (velocity zero in both X and Y-directions) boundary condition is applied.

Also the fluid coming into the model is desired to exit with zero pressure, hence the outlet i.e. the top boundary is applied with zero pressure outlet boundary condition.

The boundary conditions are assigned (to tell the system where the inlet or outlet is or where the walls are) using gambit after the completion of desired grid generation. This ensures that the solver knows where exactly the inlet, outlet and the walls of the system are before the solver initializes. The specific values of the velocities or pressures are assigned when setting up the problem in fluent solver.

Grids or Meshes Generated: Grid/mesh generation is one of the most important step in the analysis procedure. The mesh intensity is based upon the detail required at particular regions and the physics involved. The meshed are usually quadrilateral, triangle or a combination of both. Triangular elements have been used to generate the meshes for our models.

A total of 24 cases where made each at a different positioning of the valves with case 1 starting at the fully opened position to the case 24 in which the valves are almost about to close. A finer mesh is desired in the areas in between the valves and a coarser mesh is needed in the other regions. We have also added an extension channel to the top and the bottom of the model and to mesh these regions triangle elements where used as well, these elements coarse as there is no significant change occurring in the flow physics. A coarse mesh in such areas where the flow field is not significant helps us in reducing the computation time.

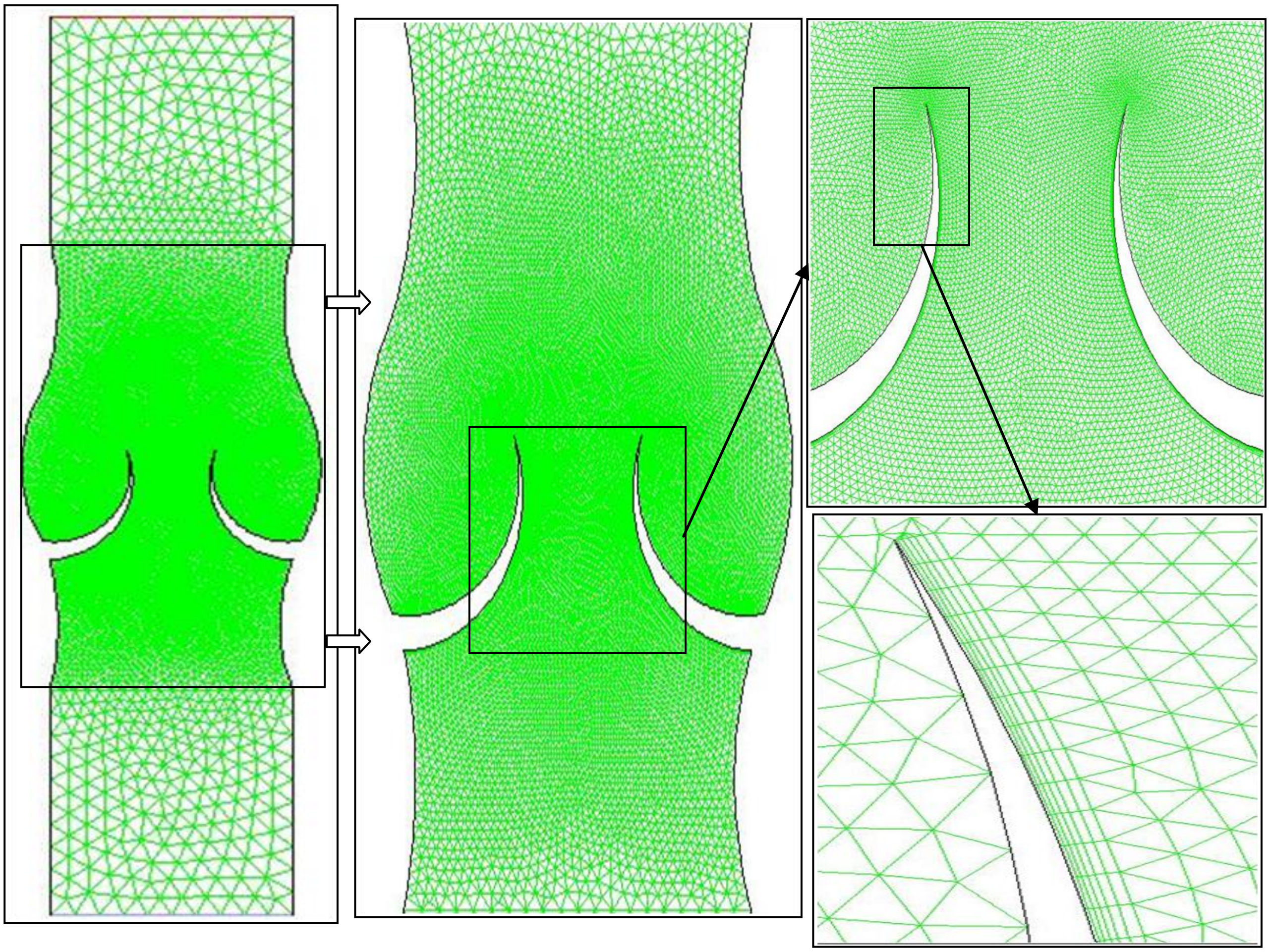

Figure 6: Grid or mesh generated for one of the cases

The above figure shows how a combination of fine and coarse elements is used in order to reduce the computation time. Also it shows the meshed extension channels and fine boundary layer elements used at the walls i.e. the valves.

One can find that a finer mesh is obtained at the areas of the valves and a coarser mesh is obtained at the extension channels, and by closer inspection we can see that the area in between the valves is given more focus by having more elements between the valves and in the pockets. The fine boundary layer elements at the valves help us to get the detail of the physics involved when fluid passes near the walls.

**3.4 Governing Equations:**

Fluid Dynamics (FD): Fluid dynamics is governed by the conservation of mass (or continuity; equation (2)) and momentum (equation (1)) principles. With continuum and incompressible fluid approximations, these principles result in partial differential equations (PDEs) and popularly known as Navier-Stokes equations (Kumar 2005).

Let $\Omega_t \subset \mathcal{R}^{n_{nd}}$ be the spatial domain with boundary $\Gamma_t$ at any instant of time $t \in (0, T)$ where $n_{sd}$ the number of is spatial dimensions (e.g., pressure and three components of velocity vectors) and $T$ is the total time of computations. The spatial coordinates and time are denoted by $\mathbf{x}$ and $t$, respectively.

*The Navier-Stokes equations of incompressible flows are*:

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Where $\rho$, $\mathbf{u}$ and $\mathbf{f}$ are the density (constant for incompressible flows), velocity, and the external force, respectively. With $\mathbf{I}$ as the identity matrix, the stress tensor ($\boldsymbol{\sigma}$) is related to the pressure (p) and shear stress tensor ($\boldsymbol{\tau}$) by

$$\rho\left(\frac{\partial \mathbf{u}}{\partial t} + \mathbf{u}.\nabla\mathbf{u} - \mathbf{f}\right) - \nabla.\boldsymbol{\sigma} = \mathbf{0} \;\; on\, \Omega_t \; \forall\, t \in (0, T), \qquad \text{EQN(1)}$$

$$\nabla.\mathbf{u} = \mathbf{0} \; on\, \Omega_t \; \forall\, t \in (0, T) \qquad \text{EQN(2)}$$

$$\boldsymbol{\sigma}(\rho, \boldsymbol{u}) = -p\boldsymbol{I} + \boldsymbol{\tau} \qquad \text{EQN(3)}$$

The shear stress ($\boldsymbol{\tau}$) for incompressible fluids is given by

$$\boldsymbol{\tau} = \begin{cases} 2\mu\dot{\boldsymbol{\varepsilon}} & \text{for Newtonian fluids i.e. } \mu = \text{constant} \\ 2\mu(\dot{\boldsymbol{\varepsilon}})\dot{\boldsymbol{\varepsilon}} & \text{for non} - \text{Newtonian fluids i.e. viscosity depends on } \dot{\boldsymbol{\varepsilon}} \end{cases}$$

Where strain rate tensor ($\dot{\boldsymbol{\varepsilon}}$) of fluid is given by

$$\dot{\boldsymbol{\varepsilon}}(\mathbf{u}) = \frac{1}{2}\left((\nabla\mathbf{u}) + (\nabla\mathbf{u})^{T}\right) \qquad \text{EQN(4)}$$

Various relationships for the viscosity approximation have reported in the literatures for the non-Newtonian fluids (e.g. "power-law" model – Ostwald & deWaele, Carreau-Yasuda model, Bingham model, Casson model, etc.).

In order to solve the equations (1) & (2), we require both prescribed velocity conditions - ($\mathbf{u} = \mathbf{g}$ on $(\Gamma_t)_g$, e.g. at the surface of arteries where velocities ($\mathbf{g}$) are prescribed by the deformation of the arteries) and flux conditions – ($n.\boldsymbol{\sigma} = \mathbf{h}$ on$(\Gamma_t)_h$, e.g. outlet where stress free conditions, i.e. **h**=0, are good approximations) boundary conditions (BCs). The initial condition for the computation was assumed equal to inlet velocity ($u(x, t = 0) = u_0(x)$).

*Problem definition and parameters*: The deep veins of the human body are modeled and solved for the fluid equations in order to better understand the flow physics involved in the deep veins. The main focus is laid on the velocity distribution and the pressure variations in order to learn about the pressure differences occurring and also to see the flow recirculation's in the velocity vector profiles. Emphasis is laid flow occurring in the two pockets and the area between the two valves.

A two dimensional double precision solver is used with Fluent in order to solve the flow equations. Because the flow is incompressible the energy equation gets decoupled from the continuity and momentum equations. So, we are only solving for the mass and momentum conservation equations.

*The power law has been used in the model the non-Newtonian blood flow and the following values were assigned* (Svelta, et al. 2003):

Power law index (n) = 0.4851

Consistency index k=0.2073 kg-s^n-2/m

Reference temperature=310K

Minimum viscosity limit=0.00125 kg/m-s

Maximum viscosity limit=0.003 kg/m-s.

Each case is run for 500 time dependent iterations with a time step of 0.01 seconds and each time step is run for 20 iterations. We were able to get converged results for flows in all the cases in 2D models. The results of these models are discussed in the following chapter.

# Chapter 4: Results and Discussions

This Chapter discusses about the physics captured by creating the vein models and running the simulations using Fluent, a commercial CFD solver. I focus on studying and analyzing the velocity profiles and the pressure contours.

In section 4.1, a two dimensional computational analysis is done on the deep veins. The cases are named from case1 to case 24 where the valves are very close to each other. These 24 cases are grouped into three different categories – fully opened, partially opened and closing groups based on the distance between the valves. The total diameter (D1) of the valve is however fixed to be 3.45mm while the distance between the valves (D2) has varied from one case to the other.

Table 1: Table showing the grouping of cases based on the distance between the

| Case no | D2/D1 | Group |
|---|---|---|
| 1 | 0.31 | Fully Opened |
| 2 | 0.30 | Fully Opened |
| 3 | 0.29 | Fully Opened |
| 4 | 0.28 | Fully Opened |
| 5 | 0.27 | Fully Opened |
| 6 | 0.25 | Fully Opened |
| 7 | 0.24 | Fully Opened |
| 8 | 0.23 | Fully Opened |
| 9 | 0.21 | Partially Opened |
| 10 | 0.20 | Partially Opened |
| 11 | 0.19 | Partially Opened |
| 12 | 0.17 | Partially Opened |
| 13 | 0.16 | Partially Opened |
| 14 | 0.15 | Partially Opened |
| 15 | 0.13 | Partially Opened |
| 16 | 0.12 | Partially Opened |
| 17 | 0.11 | Closing |
| 18 | 0.10 | Closing |
| 19 | 0.08 | Closing |

| | | |
|---|---|---|
| 20 | 0.07 | Closing |
| 21 | 0.06 | Closing |
| 22 | 0.04 | Closing |
| 23 | 0.03 | Closing |
| 24 | 0.02 | Closing |

The velocity profiles, the pressure contours, the velocity vectors and the plot of velocity vectors colored by pressure for each case are captured and some tables where created to analyze the results. These tables give us an interface to see how each of the character varies with the cases. In the velocity profiles, the dark red color represents the maximum value of velocity while the dark ink blue kind of color represents the minimum value of the velocities which is zero in most of the cases.

A more detail of the exact values can be found in the appendix where in the scale with exact values is shown for each of the cases. Similarly the colors in the pressure contours in red represent the high pressures and the ones represented by the blue color pertain to the low values of the pressure. The values of the contours vary with red being the maximum followed by orange, yellow, green and blue being the minimum for both velocities and pressures, also the respective values of the colors are shown beside them. The color bars (colored scales) seen beside the velocity contours are in meters per second and the ones next to the pressure contours are in Pascal's.

## 4.1 Two Dimensional (2D) model results:

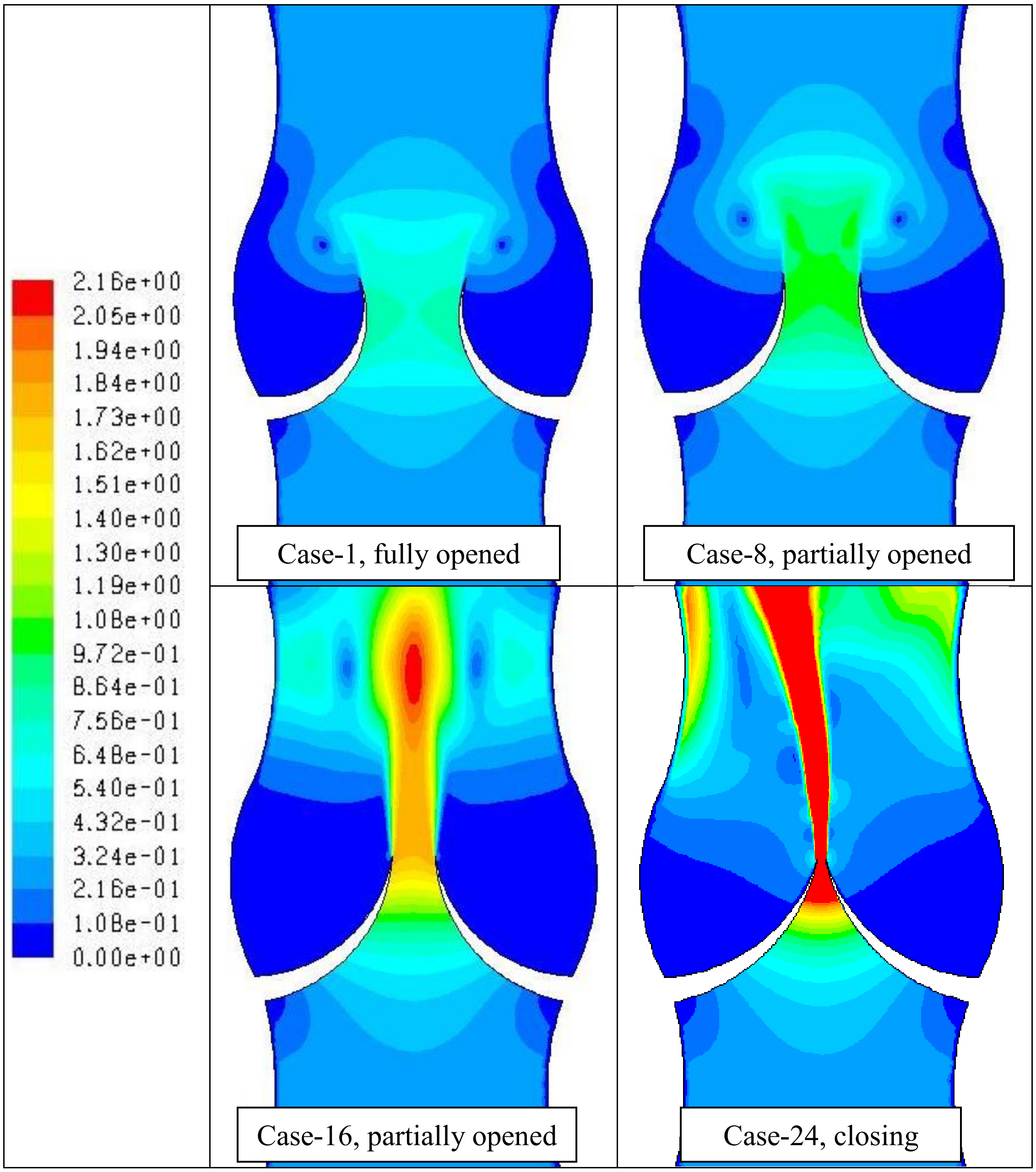



Figure 7: Velocity contours plotted for four selected cases

The above table shows the contours of the velocity and pressure within a fixed range, this helps us see the variation in the velocities and pressures from one case to the other. We can see the maximum value of the velocity in case 16 and as we go on to case 24 we find a white region which shows that the velocity in case 24 is out of bound i.e. more than the maximum velocity. This illustrates that the velocity increases as we move from case 1 to case 24 due to the decrease in the area between the valves.

One can see the light blue region developing as we progress from case 1 and case 8, into red which is maximum at case 16 and finally to white in case 24 which is out of the scaled range. Also an important thing to be observed in these cases is even though the maximum velocity changes drastically from case 1 to case 24, the pockets (or the regions between the valves and the walls) have zero velocity almost irrespective of the cases.

*Pressure Contours:*

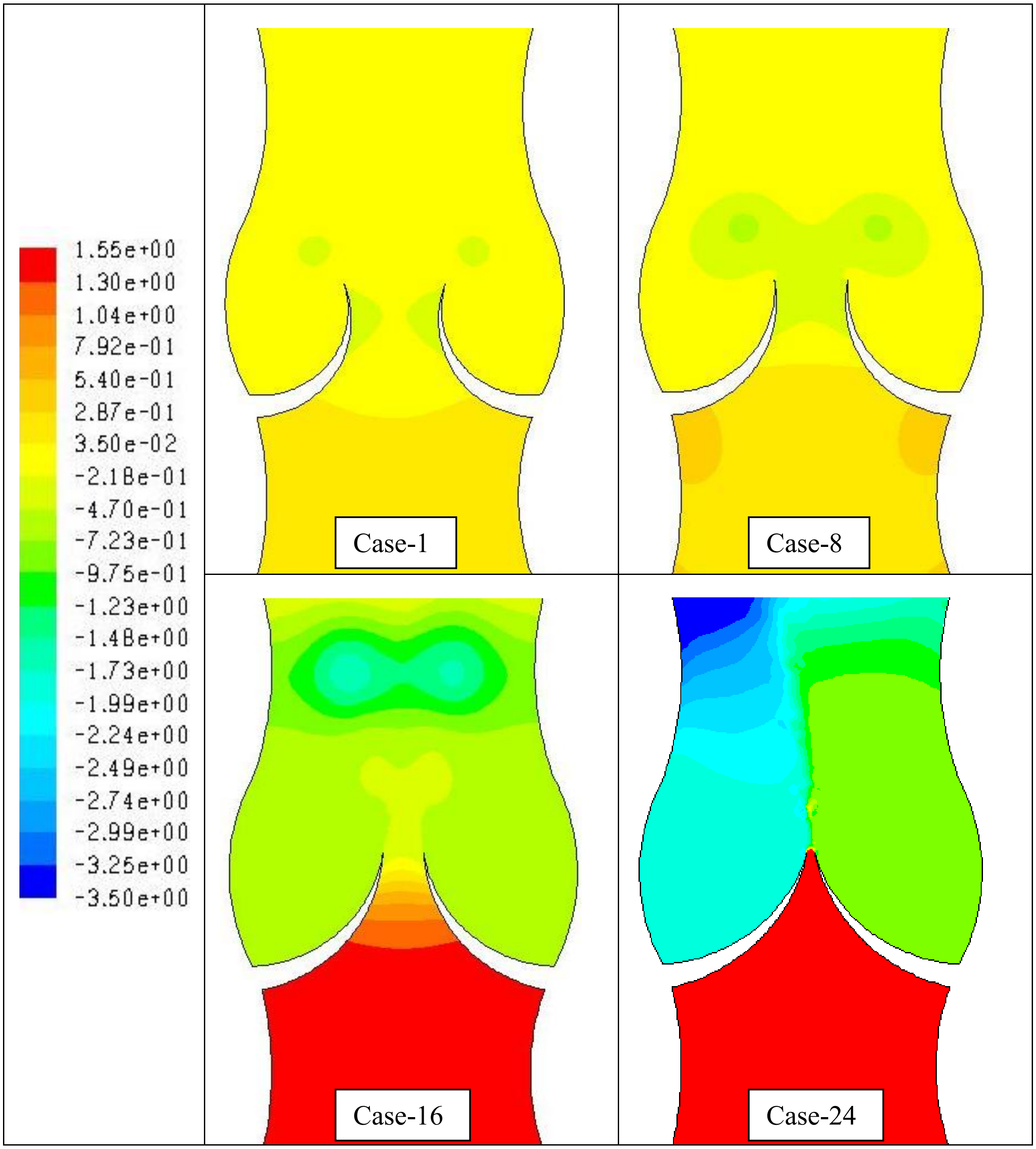


Figure 8: Pressure Contours plotted for 4 cases

The pressure contours plotted the variation of the pressures from one case to the other and also the pressure difference between the regions above and below the valves. As we see the pressure varies a lot from one case to the other, one important observation made is that the pressure behind the valves increases as the area between the valves decreases, hence we see a white region which is out of the plotted scale in case 24.

*Graphs plotted based on 2D results:*

A line was made from the center of the valves to the veins walls, and the velocity values on that line where plotted with respect to the position. A total of seven cases where picked and the velocities on the line joining the center and the vein walls (line y=1.2) were plotted for each of the cases, and a graph was plotted.

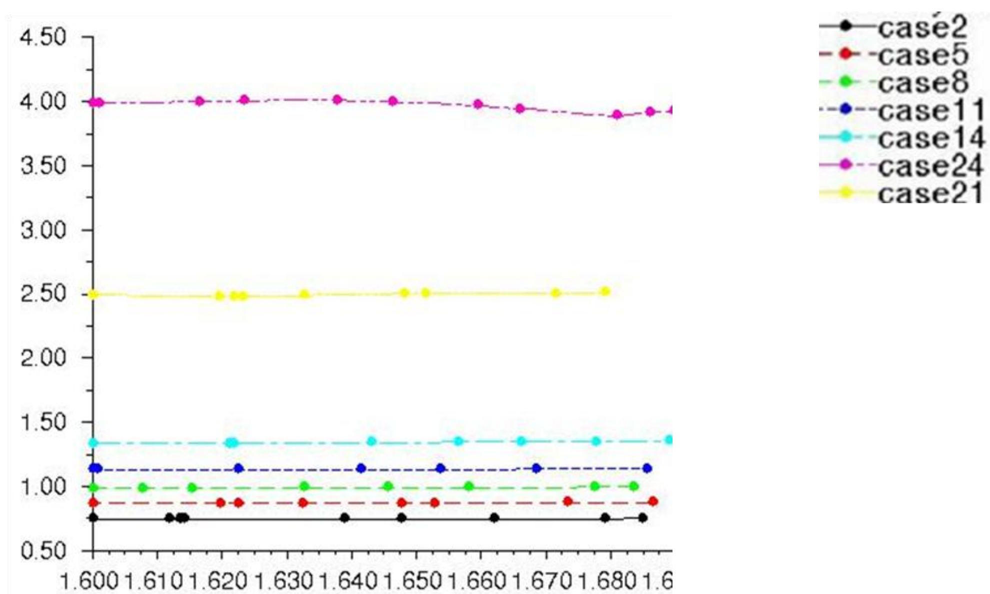



Figure 9: Velocity vs. Position at the center

In order to better understand the velocities in the pocket, the velocities and the pressures on the line plotted on the pockets were obtained.

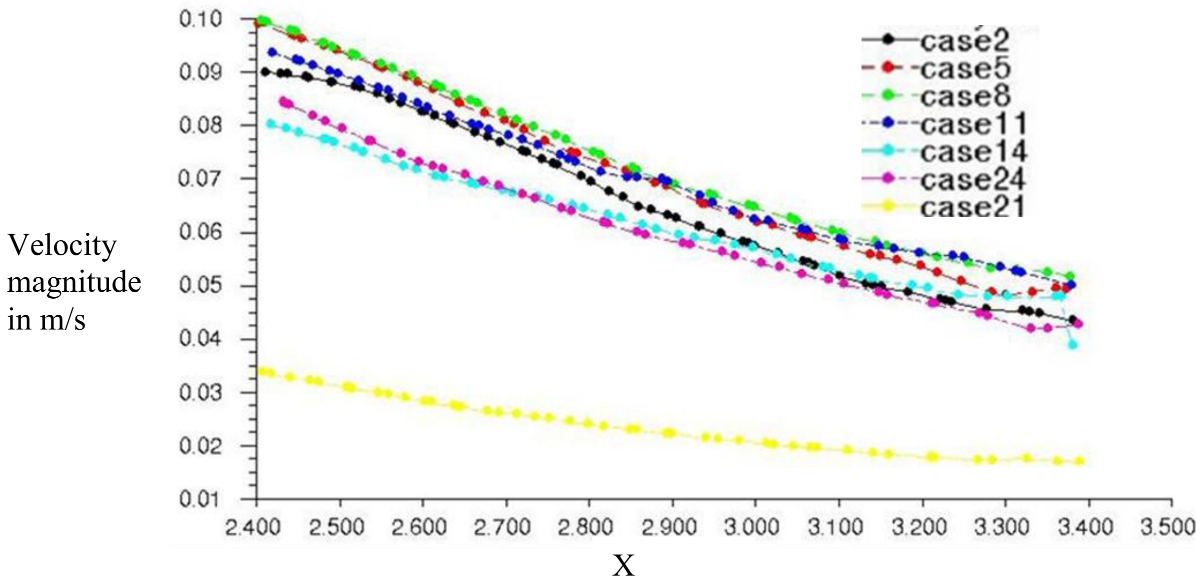



Figure 10: Velocity vs. Position in the pockets

The above graph shows that the velocity in the regions of the pockets always tends to reduce, also as the area between the valves decreases the velocity tends to further decrease in these pockets. Also not only is there an initial decrease in the velocities from case to case but also we can see that the velocities are further decreasing as we progress towards the vein valves.

Similarly the pressures were plotted for all the cases at the center and in the pockets.

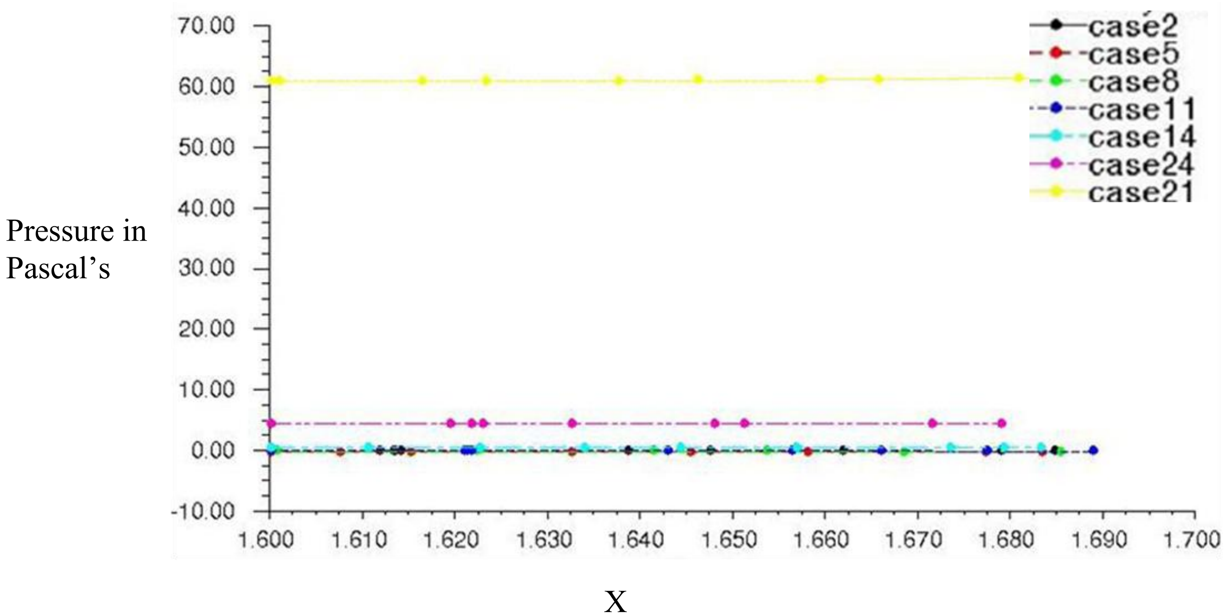



Figure 11: Pressure vs. Position at the center

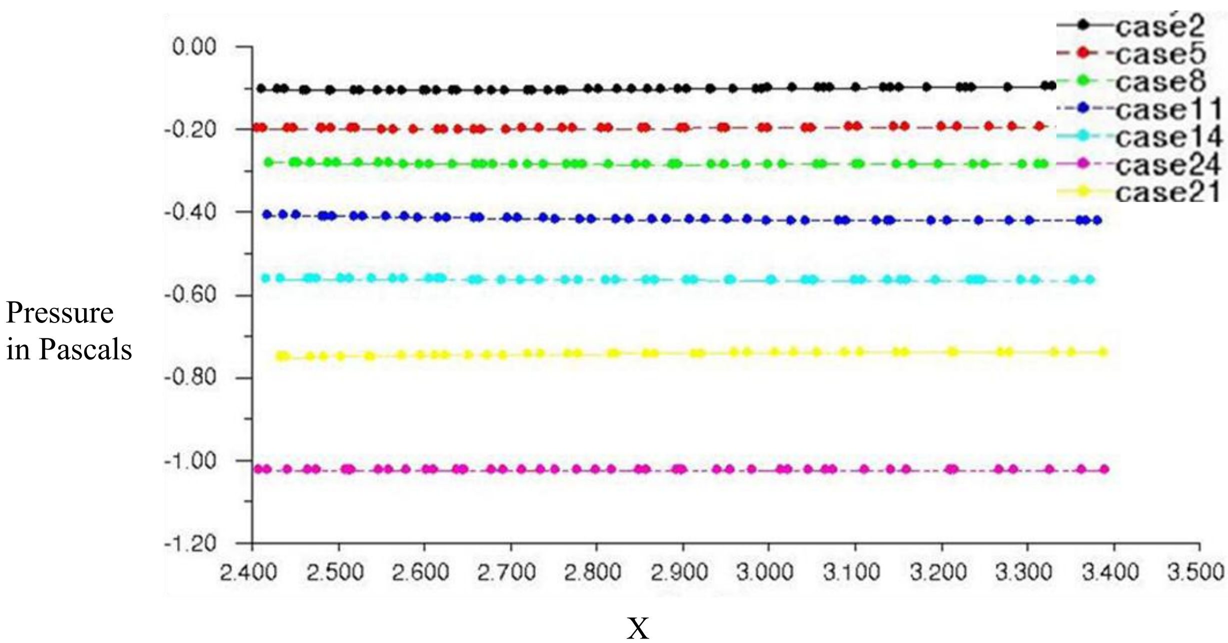
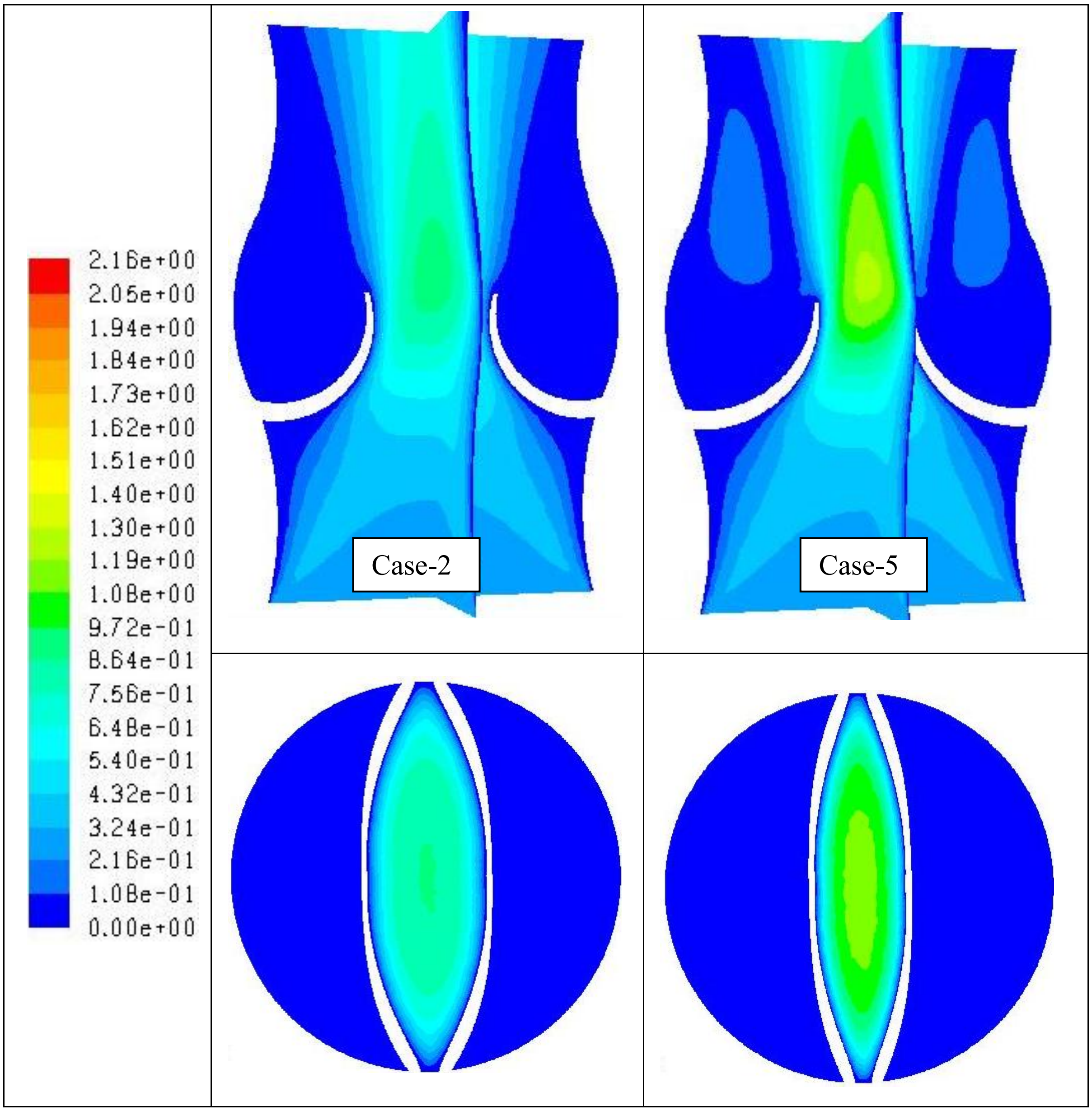



Figure 12: Pressure vs. Position in the pocket

The graphs show that the pressure is always positive or zero in most of the cases in the center and increases when the valves are coming closer to each other, but on the other hand, in the pockets the pressure is always negative (with respect to atmosphere) and tends to drop excessively as the valves approach each other. One can predict that it is this pressure difference in the pressures between the two regions which makes the valves function.

## 4.2 Three Dimensional (3D) Analysis:

*Velocity contours:*



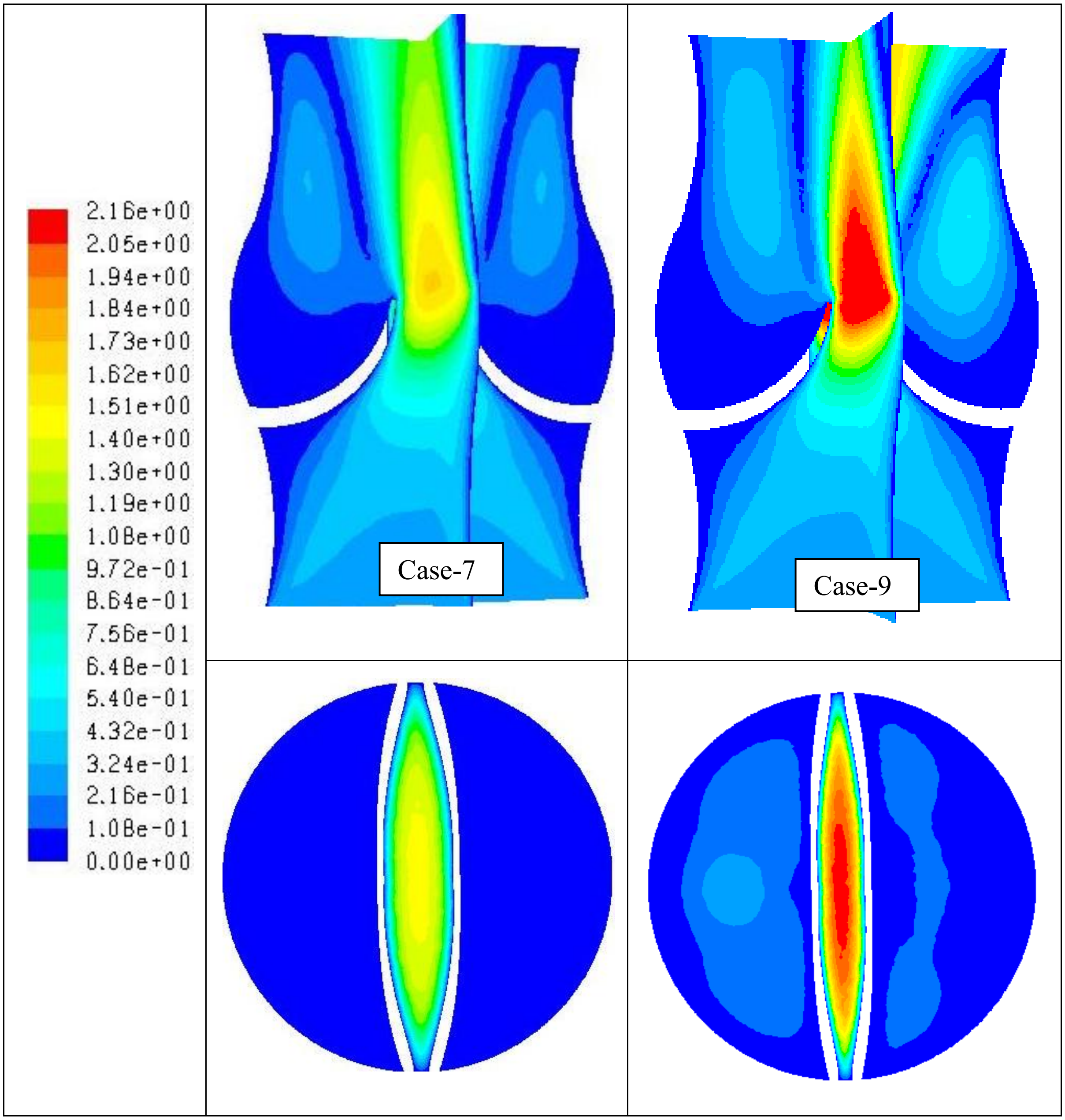


Figure 13: Velocity contours of 3D cases

A total of eight cases where constructed similarly in 3D and the fully developed velocity profiles for each of the cases are captured. The above table shows the scaled velocity which is same as the velocity range in the 2D cases and even the 3D cases show similar kind of behavior as we can see the flow to be developing into the maximum range as we move from case 2 to case 9.

The bottom picture for each of the cases shows the plane which is cut in the centre of the model and velocity contours are plotted on that plane, these picture show the top view and show a new prospective of area between the valves. In both 2D and 3D the velocities and pressures exhibit same kind of features. The reason for selecting a scaling range is to see how the maximum values develop as the valves try to close. The velocity contours are plotted on two planes created which are perpendicular to each other.

*Pressure Contours:*

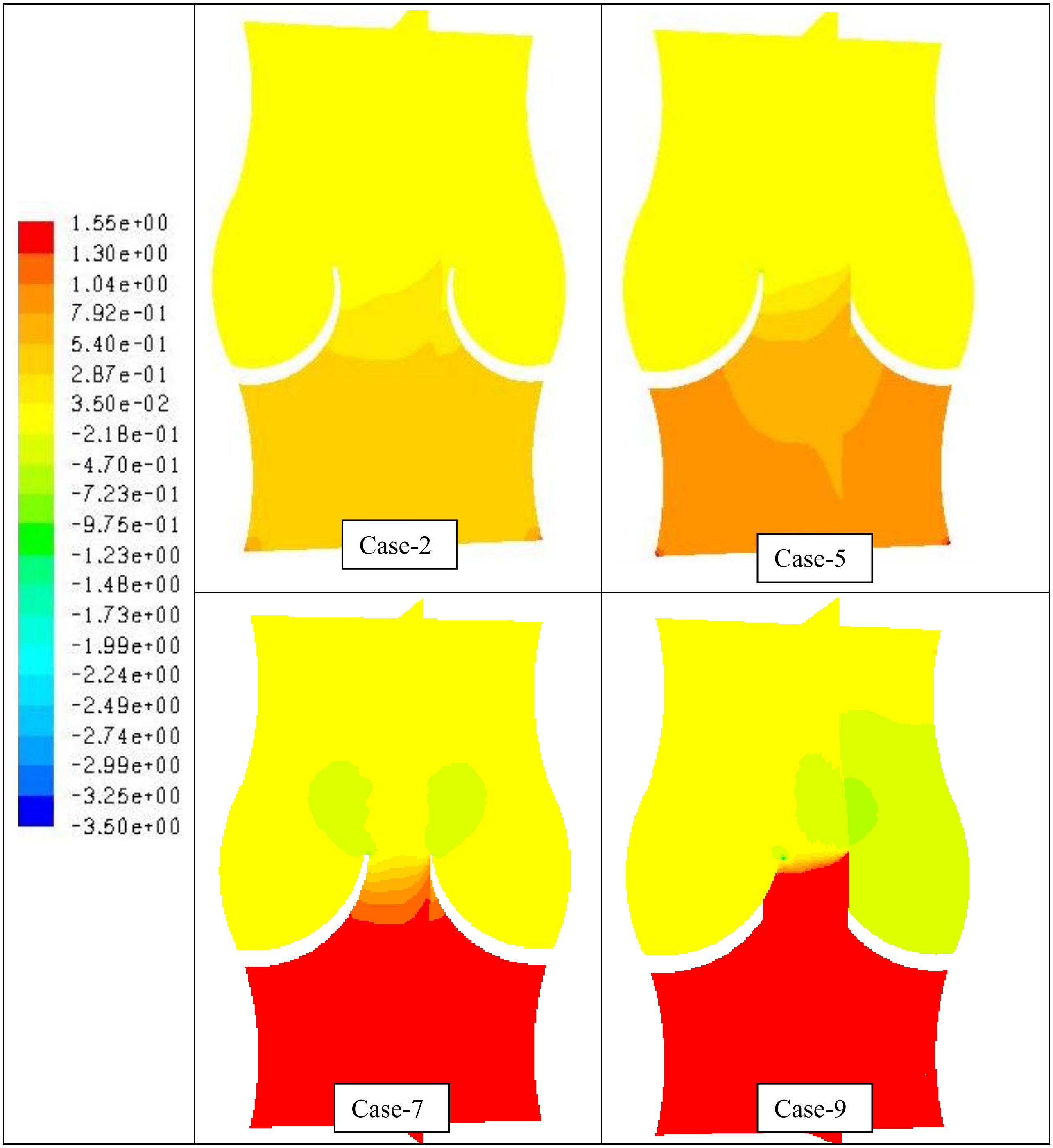



Figure 14: Pressure contours of 3D cases

The scaled pressure contours of 3D cases show the variation of pressure on a scale which is the same as the one shown in the 2D cases, this shows that the pressure variations in each cases. The contours were taken on two planes which are perpendicular to each other. The pressure contours were also plotted on two perpendicular planes that where created, the discrepancies seen especially in the final case is just because of this plotting.

The pressure builds up gradually as we move from case1 to case9; the values show that maximum pressure is seen in case 9. Also the pressure in the regions above the valves in the pockets is mostly negative.

# Chapter 5: Conclusions

We observe that the larger the opening of the venous valve, the smaller the flow acceleration through the valve. Vortices are formed after crossing the valves at different places for different opening-closing cases of the valves. Vortices are noticed in the velocity profile depicting circular profile and also in the pressure profiles with minimum pressure at the center of a circulating region. We believe that the areas where vortices are trapped in the pockets with low velocities or the areas with almost stagnant velocities are more susceptible to clot formation. These areas are mainly the areas between vein valves and the vein wall.

One can observe that the pressures in the regions behind the valves and in the regions of the pockets are never the same (in most of the cases the pressures are maximum at the bottom and almost negative in the pockets); it is this pressure difference between the two regions which allows the blood to flow and the valves to open when the muscles contract and squeeze the veins.

We can observe that there is always a pressure being build up in the regions behind the valves, this is due to the reducing area between the valves, but if observed carefully we can see that the variations of pressure is more at the regions nearing the tips of the valves. One can say that it is these pressure variations, which intern are the cause of fluttering kind of phenomena seen in the valves when there is blood flowing through them.

# Chapter6: Future Work

An experiment is being setup at the lab, in order to measure velocities and pressures of the venous valves. Experimental subjects with different geometries will be used in the place of veins, the setup simulates flow environment of the deep veins. Cameras are going to be used to capture the motion of the particles which will in turn be illuminated by the Particle Image Velocimetry (PIV) system. A motor is used to pump the fluid to the system and pressure transducers are going to be used to measure the drops in pressure of the experimental subjects. Glycerin and sodium chloride solutions are going to be used as the experimental fluid, because of their physical properties.

Figure 15: Experimental setup being installed

# Bibliography

Abbade, Luciana. P. Fernandes, and Sidnei Lastoria. "Venous Ulcer: epidemology, physiopathology, diagnosis and treatment." *International Society of Dermatology* (International Journal of Dermatology) 44 (2005): 449-456.

Alberto, Figueroa c, Vignon-Clementel E Irene, Jansen E Kenneth, Hughes J.R. Thomas, and Taylor A Charles. "A coupled momentum method for modeling blood flow in three-dimensional deformable arteries." *Computer methods in applied mechanics and engineering*, 2006: 5685-5706.

Anne, Aquila M. "Deep Venous Thrombosis." *Journal of Cardiovascular Nursing*, July 2001: 25-44.

Anthonie, Lensing WA, Pradoni Paolo, Prins H Martin, and R H Buller. "Deep-vein thrombosis SEMINAR." *The Lancet*, 1999.

Bader, Hermann. "The Anatomy and physiology of the vascular wall." In *Handbook of Physiology ~ circulationII*.

Bertina, Rogier M. "The role of procoagulants and anti coagulants in the development of venous thromboembolism." *Thrombosis Research* (Thrombosis Research), 2009: S41-S45.

Buescher, C. D, B Nachiappan, J. M Brumbaugh, K. A Hoo, and H. F Janssen. "Experimental Studies of the Effects of Abnormal Venous Valves on fluid flow." (American Chemical Society and American Institute of Chemical Engineers) 2005.

Clagett, G.Patrick, Fredrick.A Anderson Jr, Mark. N Levine, Edwin.W Salzman, and H. Brownell Wheeler. "Prevention of venous Thromboembolism." *CHEST: official publication of the American College of Chest Physicians* (CHEST official publication of the American college of chest physicians), 1992: 391s-407s.

Clagett, G.Patrick, Fredrick.A Anderson Jr, Mark. N Levine, Edwin.W Salzman, and H. Brownell Wheeler. "Prevention of venous Thromboembolism." *CHEST: official publication of the American College of Chest Physicians*, 1992: 391s-407s.

Coon, William W. "Epidemology of Venous Thromboembolism." *Annals of Surgery*, 1977: 149-160.

David, Steinman A. "Image-Based Computational Fluid Dynamics Modeling in Realistic Arterial geometries." *Biomedical Engineering Society*, 2002: 483-497.

David, Steinman A, Milner S Jaques, Norley J Chris, Lownie P Stephen, and Holdsworth W David. "Image-Based Computational Simulation of Flow Dynamics in a Gaint Intracranial Aneurysm." *American Society of Neuroradiology*, 2003: 559-566.

Donahue, Brian S. "Factor V Leiden and Perioperative Risk." *International Anesthesia Research Society*, 2004: 1623-34.

Eran, Weinmann E, and Salzman W Edwin. "Deep-Vein Thrombosis." *The New England Journal of Medicine*, 1994: 1630-1641.

Franklin, K J. "Valves in Veins: An Historical Survey." *Royal Society of Medicine*, 1927: 1-33.

Fredrick, Anderson A. Jr, and Spencer A Fredrick. "Risk Factors of Venous Thromboembolism." *Journal of The American Heart Association*, 2003.

Georgia, Pambianco, Orchard Trevor, and Landau Philip. "Deep Vein Thrombosis: Prevention in Stroke Patients During Rehabilitation." *Arch Physics Med Rehabil*, 1995: 324-331.

Gottlob, R, and R May. *Venous Valves: Morphology, Function, Radiology, Surgery.* New York: Springer-Verlag.
Heit, John.A, Catie.E Kobbervig, Andra.H James, Tanya.M Petterson, Kent. R Bailey, and L. Joeseph Melton III. "Trends in the Incidence of Venous Thromboembolism during Pregnancy or Postpartum: A 30-Year Population based study." *Annals of Internal Medicine*, 2005: 697-706.
Helps, E. P W, and D A McDonald. "Obeservations on Laminar Flow in Veins." *J.Physiol.*, 1954: 631-639.
Herbert Janssen, Christina Trevino, David Williams. "Hemodynamic Alterations in venous blood flow produced by external pneumatic compression." *The Journal of Cardiovascular Surgery*, 1993: 441-447.
Herbert, Janssen F, Schachner Jean, Hubbard Joel, and Hartman J Ted. "The Risk of Deep Venous Thrombosis: A Computerized Epidemiologic Approach." *Surgery*, 1986: 205-212.
James, Hathcock J. "Flow Effects in Coagulation and Thrombosis." *Journal of The American Heart Association*, 2006: 1729-1737.
Janseen, Herbert. F, Jean Schachner, Joel Hubbard, and J. Ted Hartman. "The risk of deep venous thrombosis: A computerized epidemiologic approach." *Surgery* (Surgery), 1986: 205-212.
Jerry, Avorn, and c. Winkelmayer Wolfgang. "Comparing the Costs, Risks and Benifits of Competing Strategies for the Primary Prevention of Venous Thromboembolism." *Journal of American Heart Association* (Journal of American Heart Association), 2004: IV25-IV32.
John, Cimbala M, and Cengel A Yunus. *Essentails of Fluid Mechanics Fundamentals and applications.* New York: The McGraw-Hill companies, 2008.
Joseph, Stanton R, Freis D Edward, and Wilkins W Robert. "The Acceleration of Linear Flow in The Deep Veins of The Lower Extremity of Man By Local Compression." 1948.
Juan, Cebral R, Yim J Peter, Lohner Rainald, Soto Orlando, and Choyke L Peter. "Blood Flow Modeling in Carotid Arteries with Computational Fluid Dynamics and MR Imaging." *Academic Radiology*, 2002: 1286-1299.
Kakkar, V V, C T Howe, A N Nicolaides, J T G Renney, and M B Clarke. "Deep Vein Thrombosis of the Leg." *American Journal of Surgery*, 1970: 527-530.
Kumar, Vinod. "Advanced Computational Techniques for Incompressible / Compressible Fluid-Structure Interactions." Rice University, 2005.
M.M.W, Koopman, Beek E.J.R Van, and Cate J W Ten. "Diagnosis of Deep Vein Thrombosis." *Progress in Cardiovascular diseases*, 1994: 1-12.
Mark, Meissner H, et al. "The hemodynamics and diagnosis of Venous disease." *Journal of Vascular Surgery*, 2007: 4s-24s.
Meyer-Michel, Samama. "An Epidemiologic Study of Risk Factors for Deep Vein Thrombosis in Medical Outpatients." *ARCH INTERN MED*, 2000: 3415-3420.
National Institues of Health. *Prevention of Venous Thrombosis and Pulmonary embolism.* Consensus Development Conference Statement, 1986.
Nurullah, Arslan, Tuzcu Volkan, Nas Selman, and Durukan Ause. "CFD modeling of blood flow inside human left coronary artery bifurcation with aneurysms." *The 3rd European Medical and Biological Engineering Conference*, 2005.
Paul, Kyrle A, and Eichinger Sabine. "Deep vein thrombosis." *The Lancet*, 2005: 1163-1174.
Philips, Wells S, et al. "Accuracy of clinical assessment of deep-vein thrombosis." *Lancet*, 1995: 1326-1330.

Robert, Kistner L. "Primary Venous Valve Incompetence of the leg." *The American joournal of Surgery*, 1980.
Stein, Paul.D, Russel. D Hull, Fadi Kayali, William.A Ghali, Andrew. K Alshab, and Ronald. E Olson. "Venous Thromboembolism According to Age: The Impact of Aging Population." *American Medical Association* (ARCH INTERN MED) 164 (2004): 2260-2265.
Svelta, Petkova, Hossain Alamgir, Naser Jamal, and Palombo Enzo. "CFD Modelling of Blood Flow in Portal Vein Hypertension with and without Thrombosis." (Third International Conference on CFD in the Minerals and Process Industries) 2003.
Wee-shian, Chan, and Ginsberg S Jeffrey. "Diagnosis of deep vein thrombosis and pulmonary embolism in pregnancy." *Thrombosis Research*, 2002: 85-91.
Wijeratne, N.S, and K.A Hoo. "An analytical approach to identify fluid flow seperation and re-attachment in a collapsible channel." *computers and chemical engineering* (computers and chemical engineering) 31 (2006): 346-360.
Wijeratne, Nilmini Saumya. "Fluid Flow Studies in Flexible Tubes with Internal Flexible Structures." 2008.
Yuchen, Qui, R C Quijano, S K Wang, and Hwang H C Ned. "Fluid Dynamics of Venous Valve Closure." *Annals of Biomedical Engineering*, 1995: 750-759.
Zaho, S Z, A D Huges, S A Thom, A V Stanton, B Ariff, and Q Long. "Blood flow and vessel mechanics in a physiologically realistic model." *Journal of Biomechanics*, 2000: 975-984.

# Appendix

Case1:

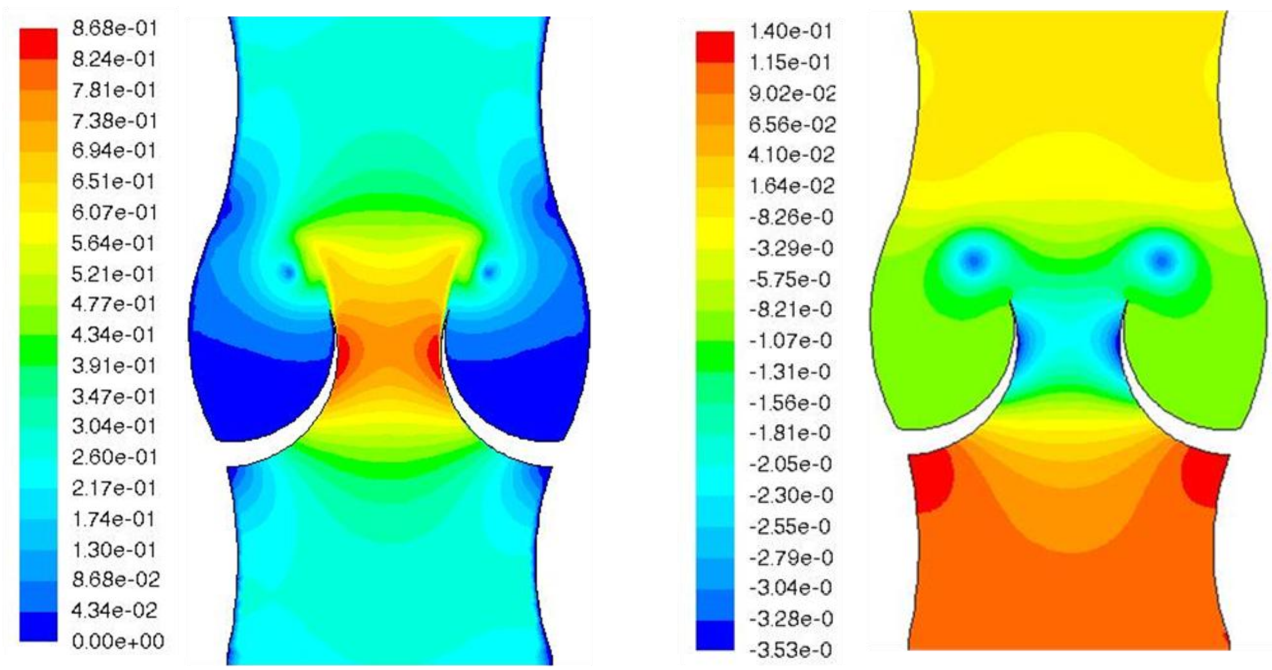



Figure 16: Velocity and Pressure Contours (case1)

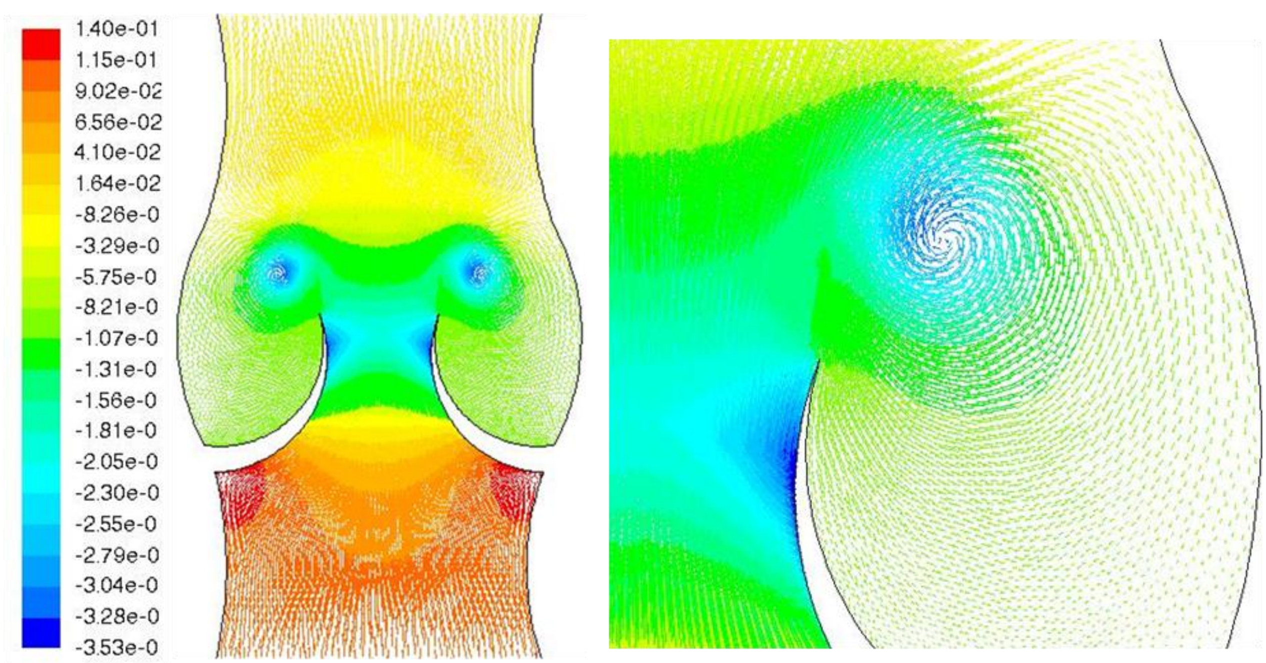



Figure 17: Velocity vectors Colored by Pressure (case1)

Case2:

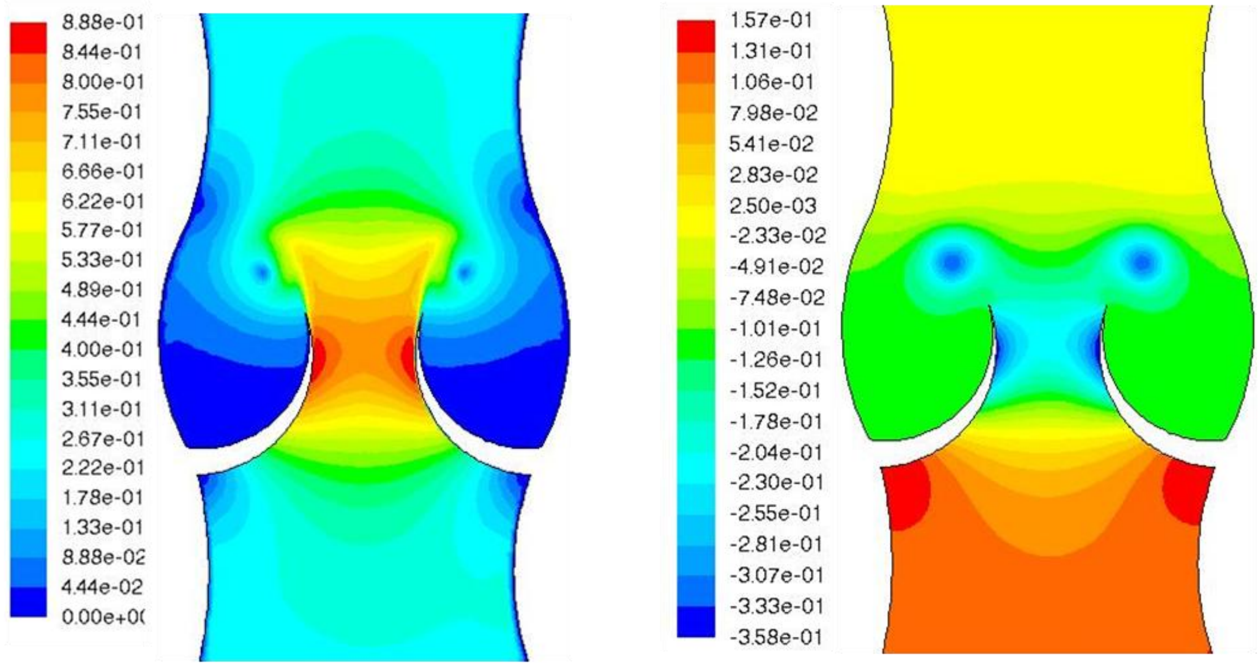



Figure 18: Velocity and Pressure contours (case2)

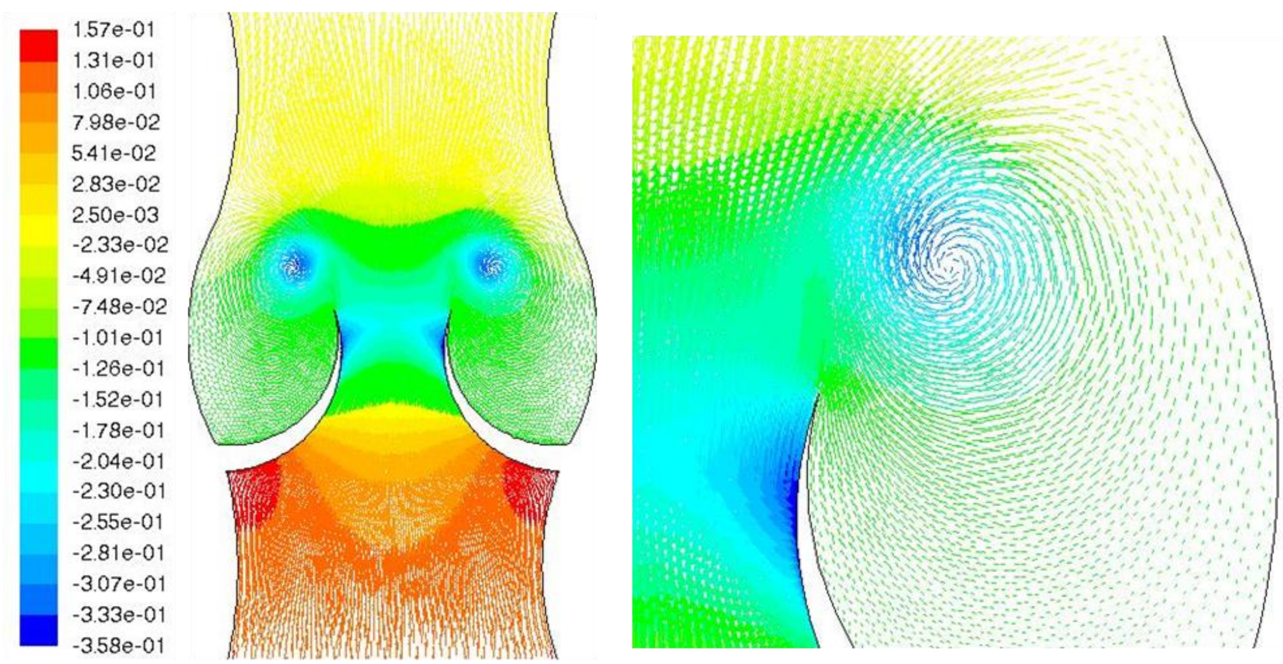



Figure 19: Velocity vectors colored with Pressure (case2)

Case3:

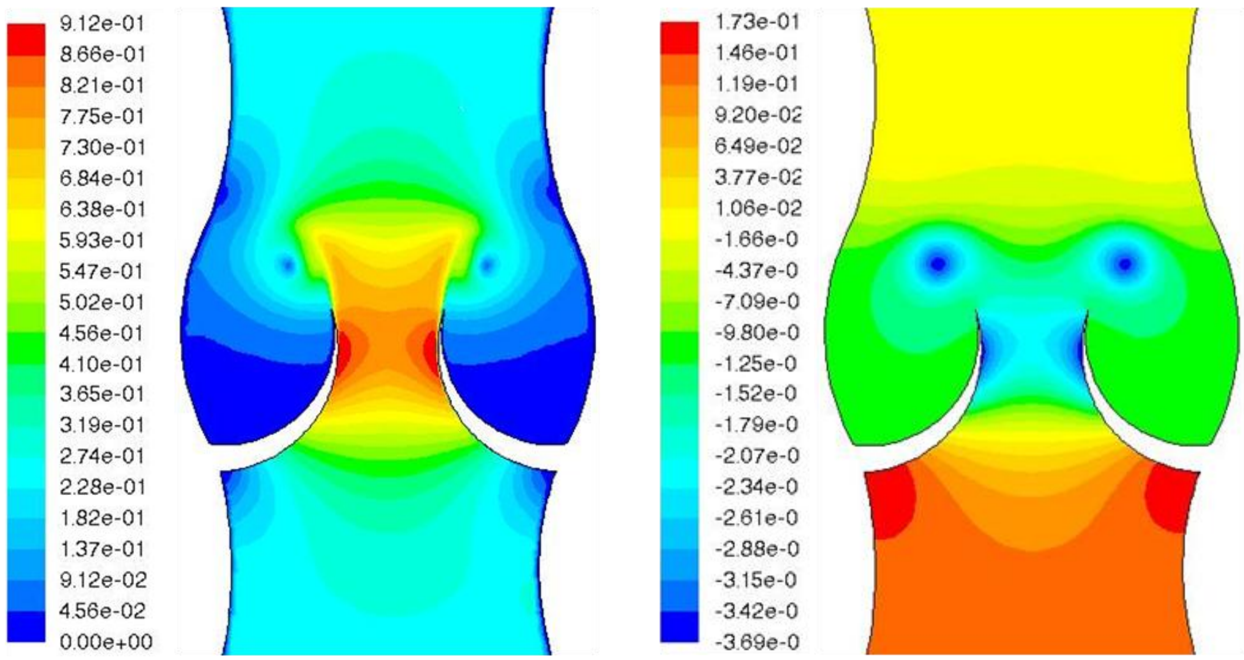



Figure 20: Velocity vectors Colored by Pressure (case3)

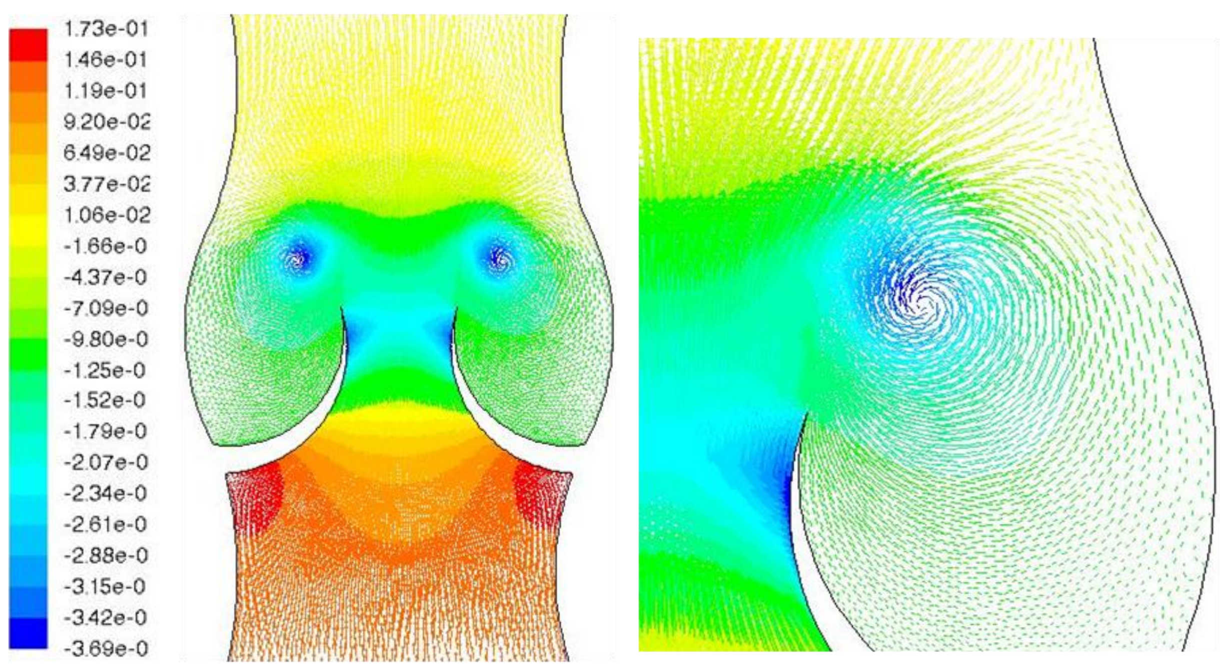



Figure 21: Velocity vectors colored with Pressure (case3)

Case4:

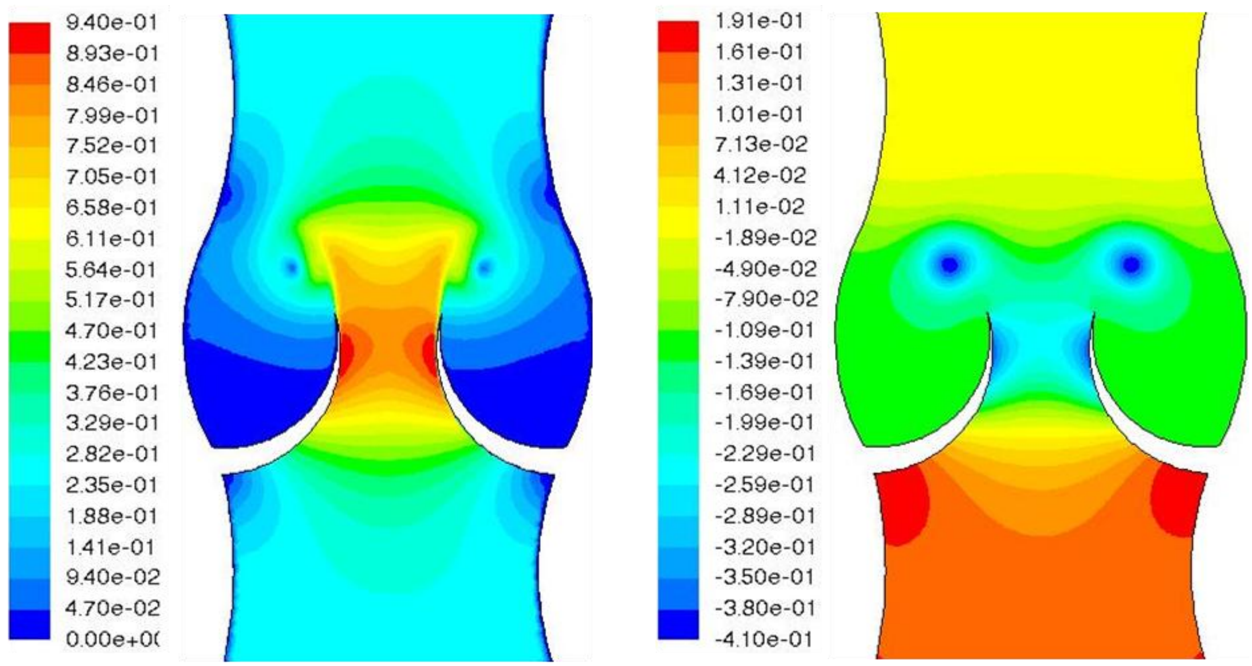



Figure 22: Velocity and Pressure Contours (case4)

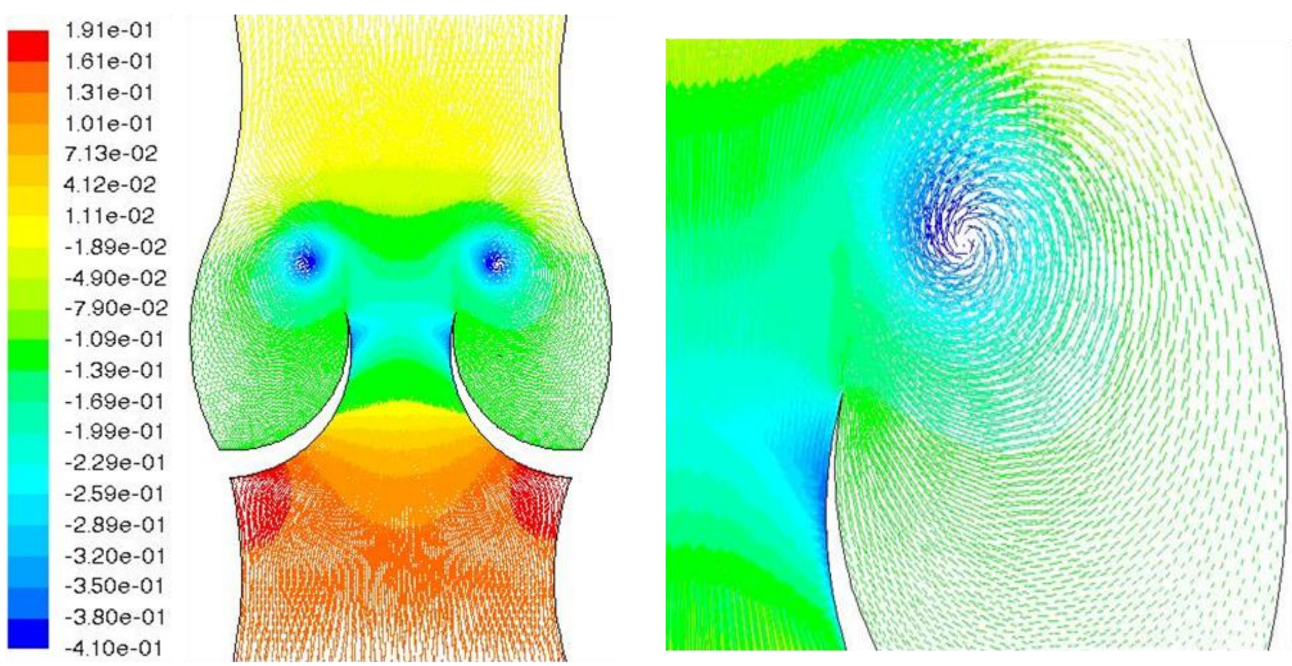



Figure 23: Velocity vectors colored with Pressure (case4)

Case5:

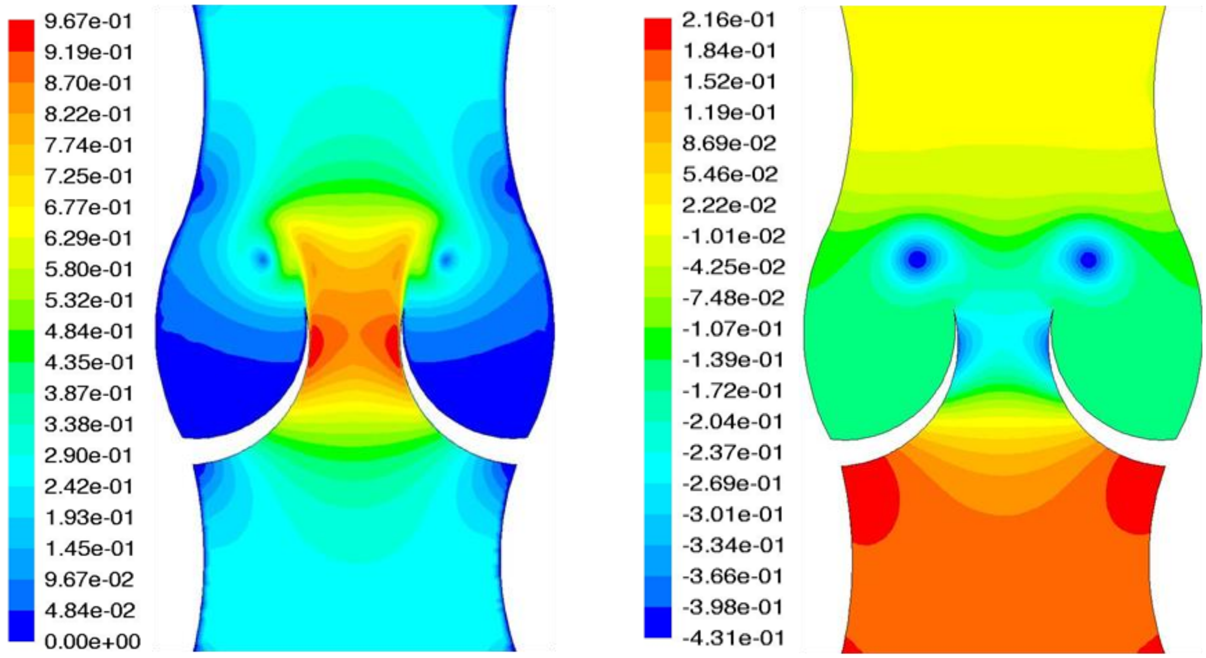



Figure 24: Velocity and Pressure Contours (case5)

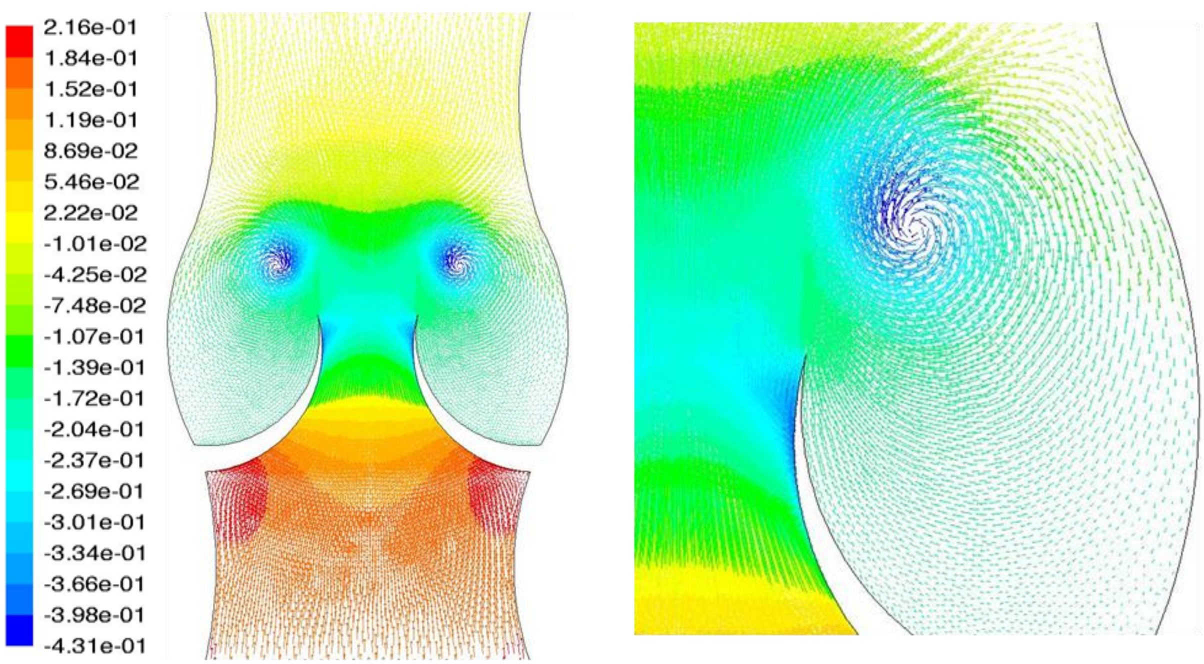



Figure 25: Velocity vectors colored with Pressure (case5)

Case6:

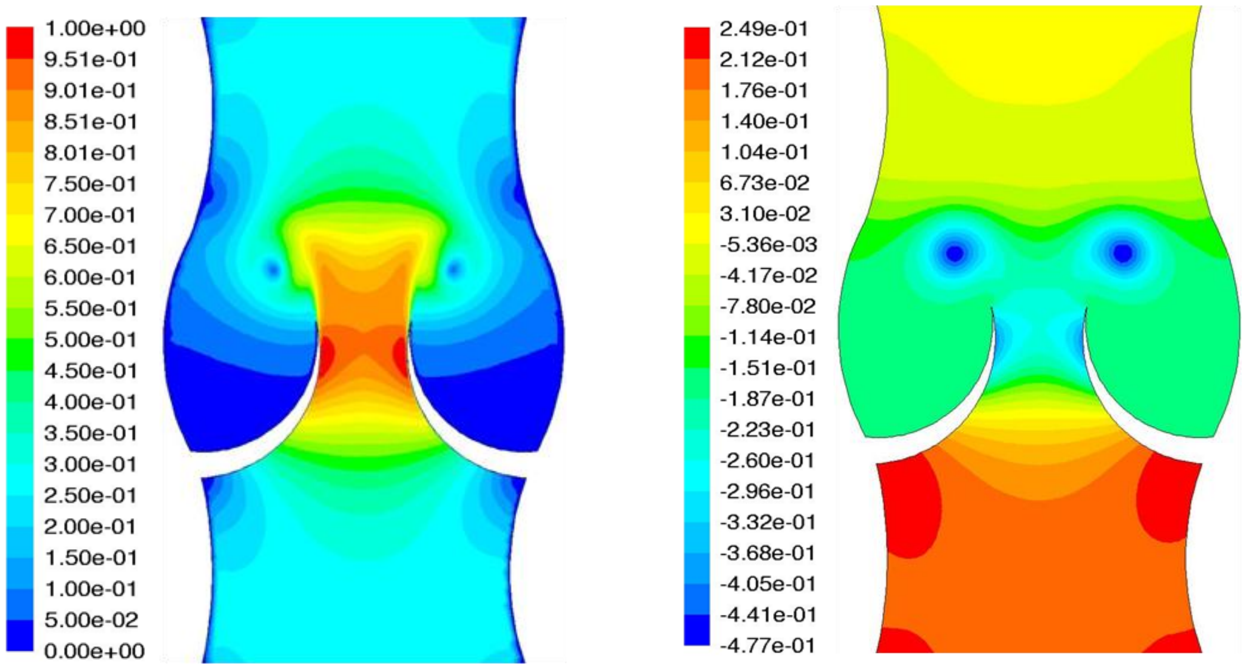



Figure 26: Velocity and Pressure Contours (case6)

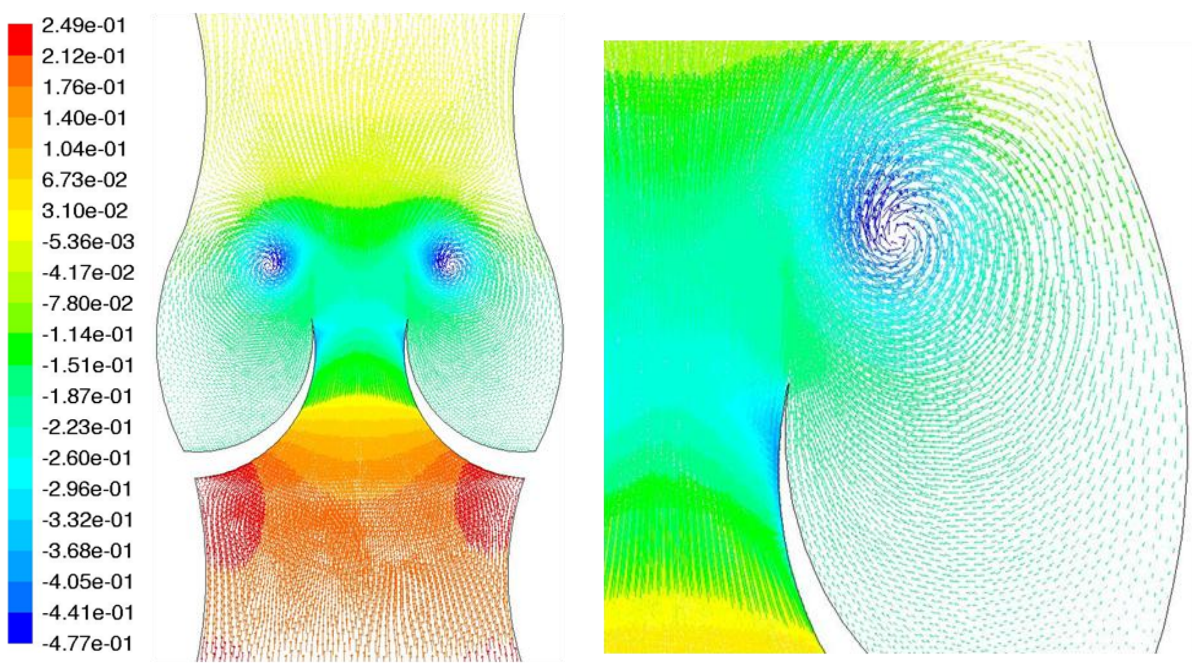



Figure 27: Velocity vectors colored with Pressure (case6)

Case7:

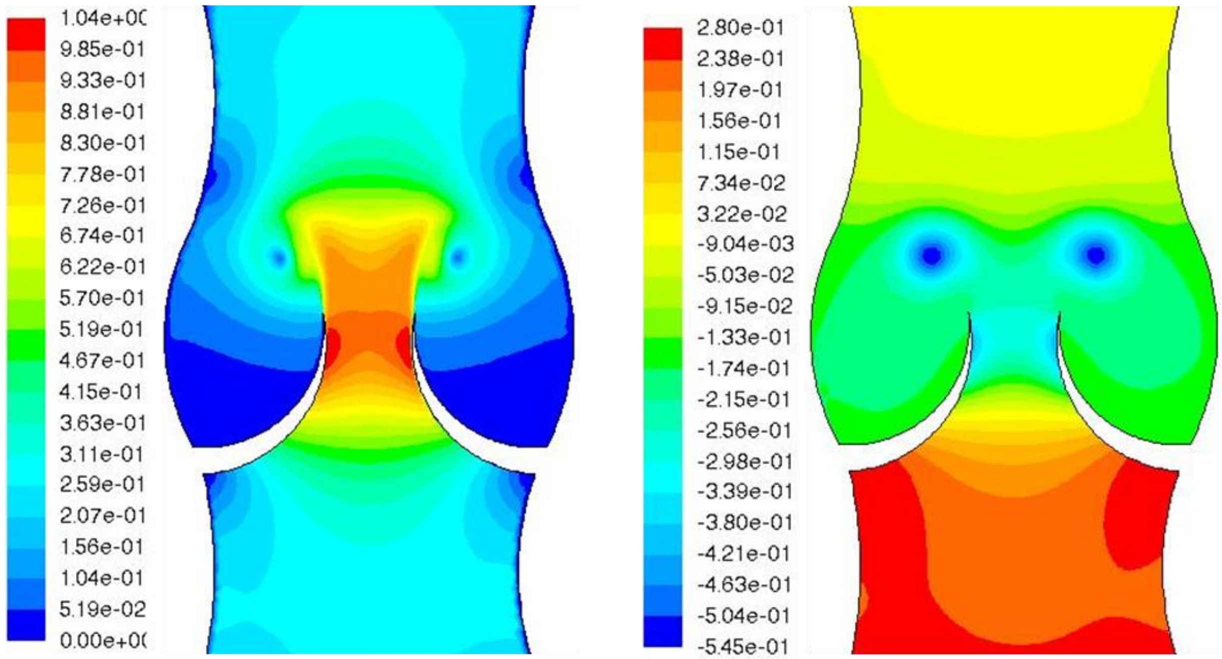



Figure 28: Velocity and Pressure Contours (case7)

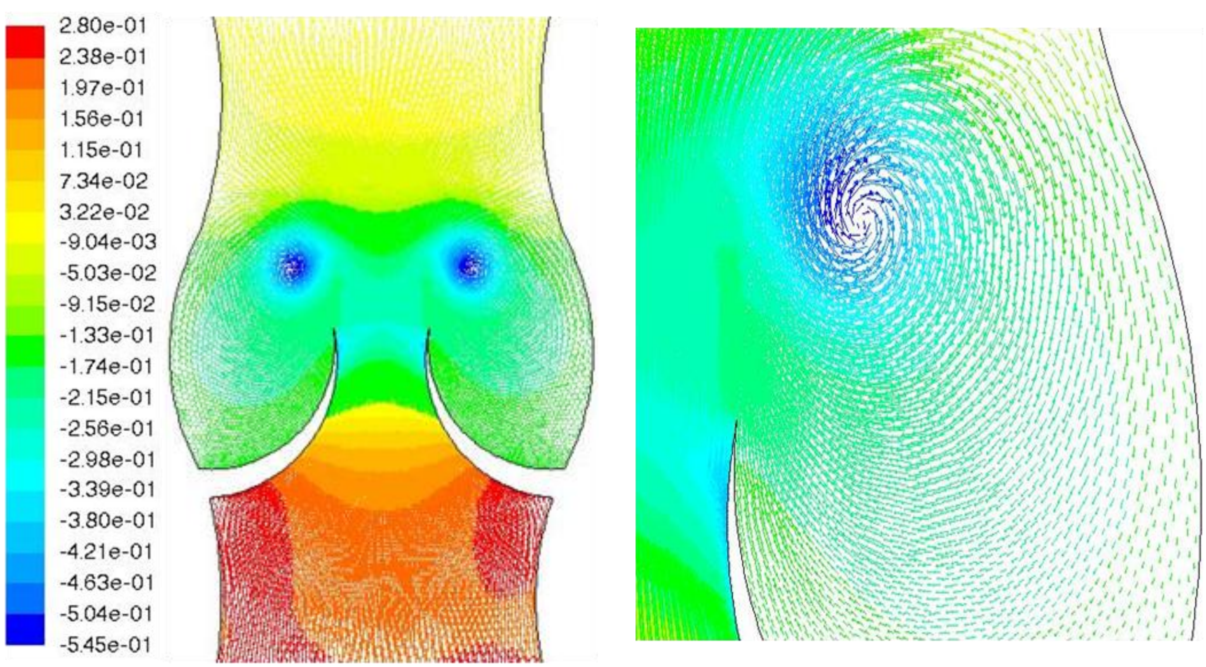



Figure 29: Velocity vectors colored with Pressure (case7)

Case8:

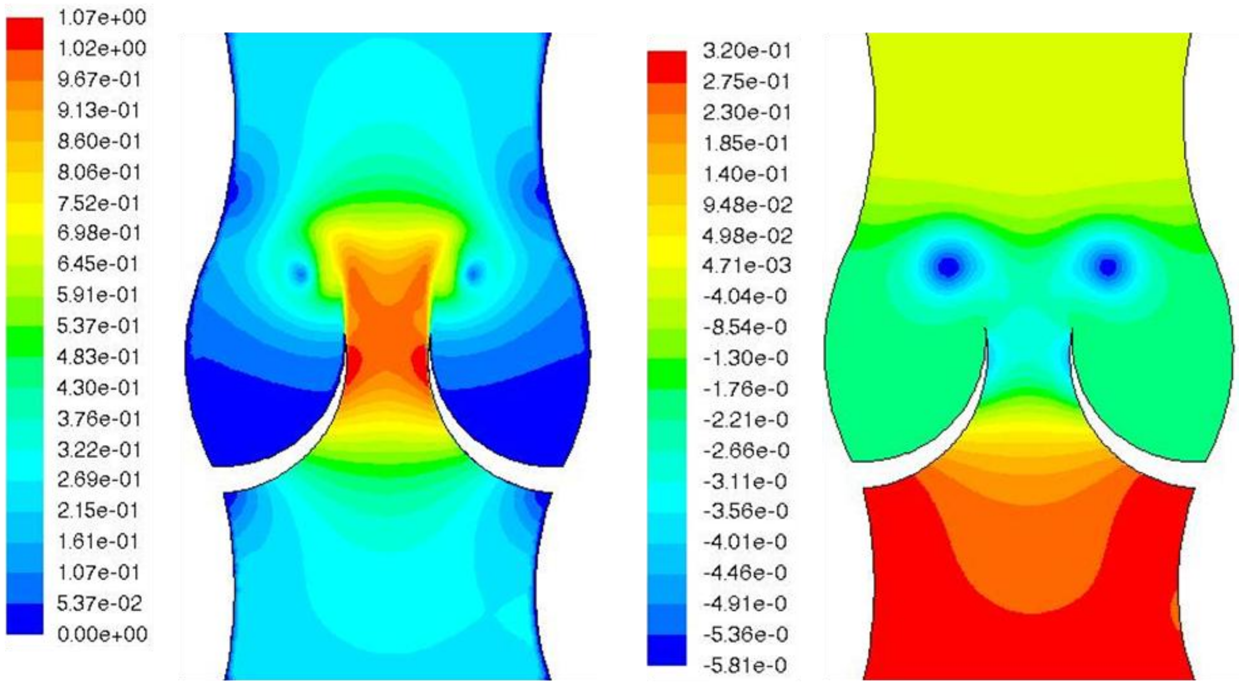



Figure 30: Velocity and Pressure Contours (case8)

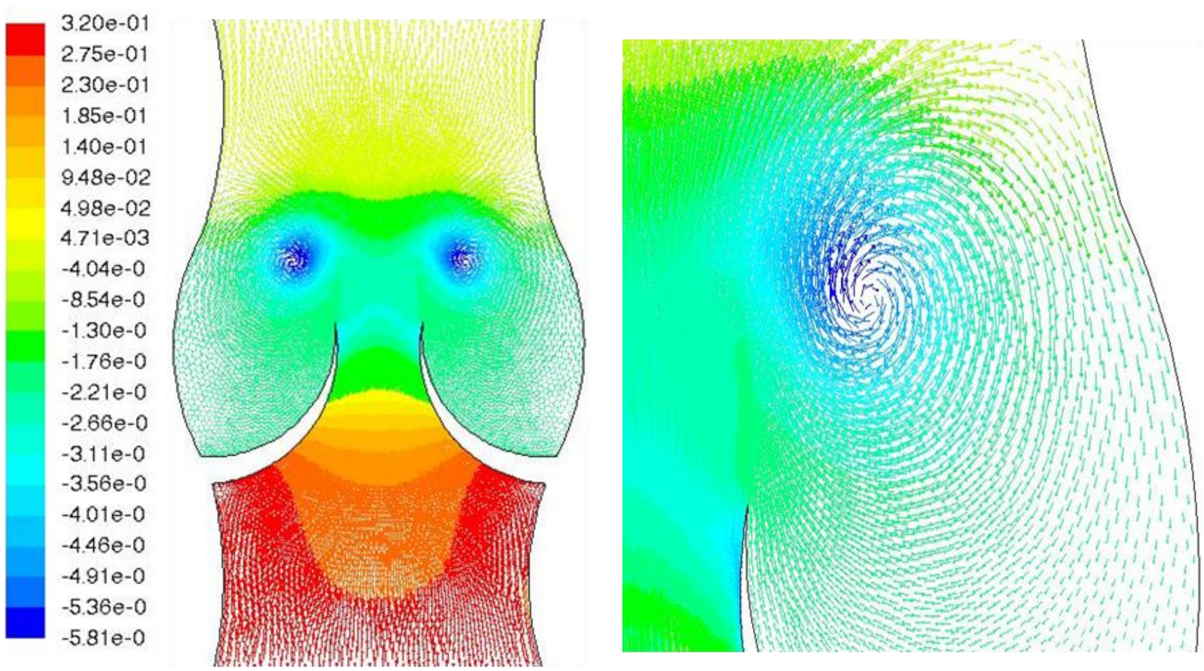



Figure 31: Velocity vectors colored with Pressure (case8)

Case9:

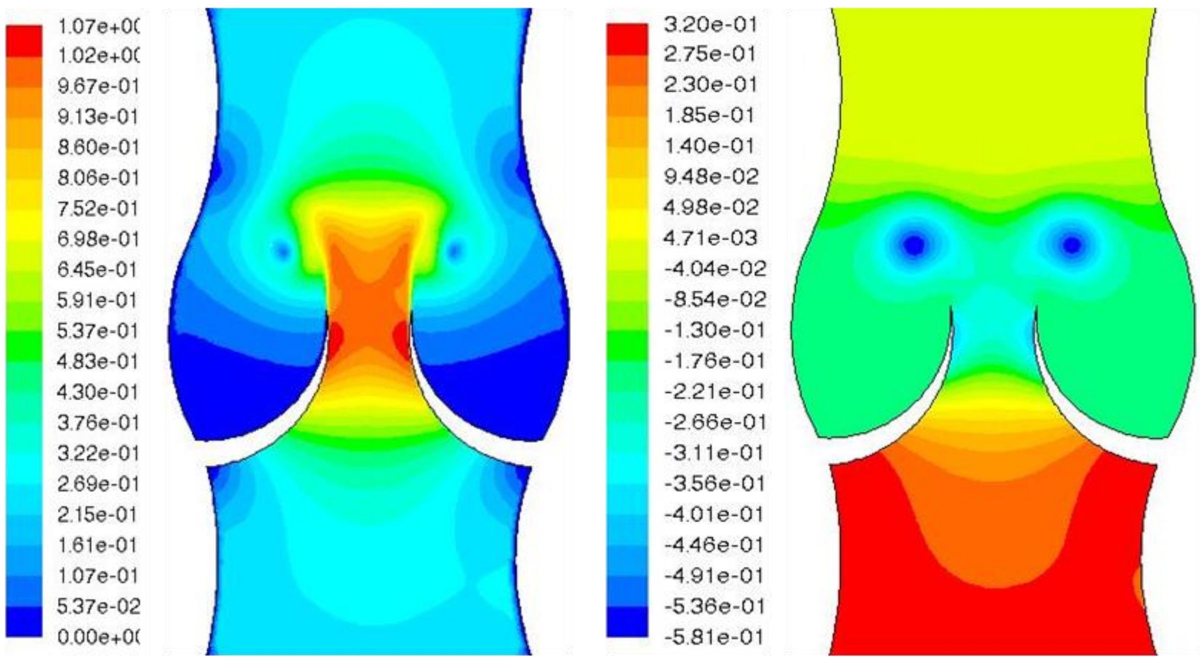



Figure 32: Velocity and Pressure Contours (case9)

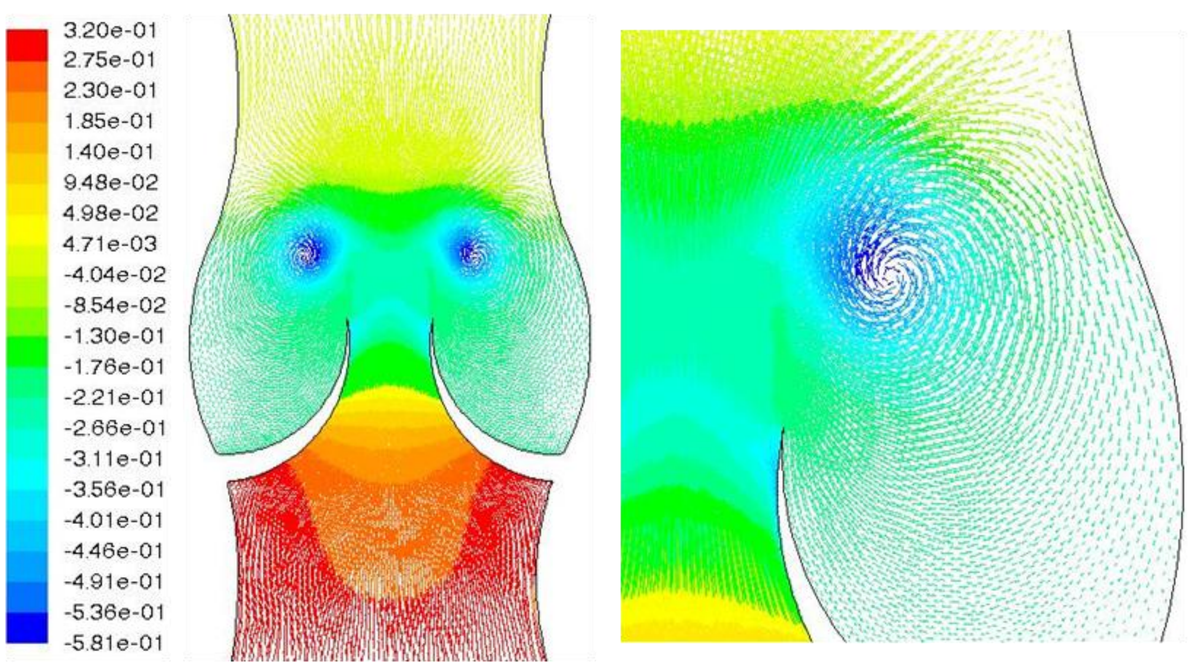



Figure 33: Velocity vectors colored with Pressure (case9)

Case10:

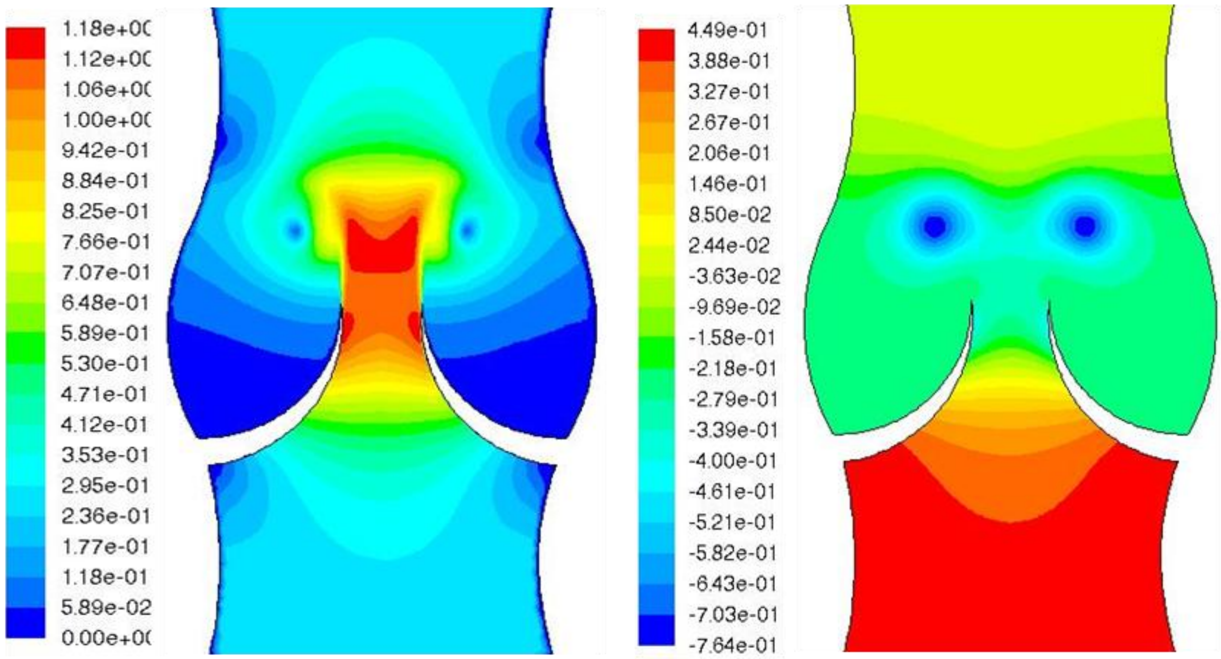


Figure 34: Velocity and Pressure Contours (case10)

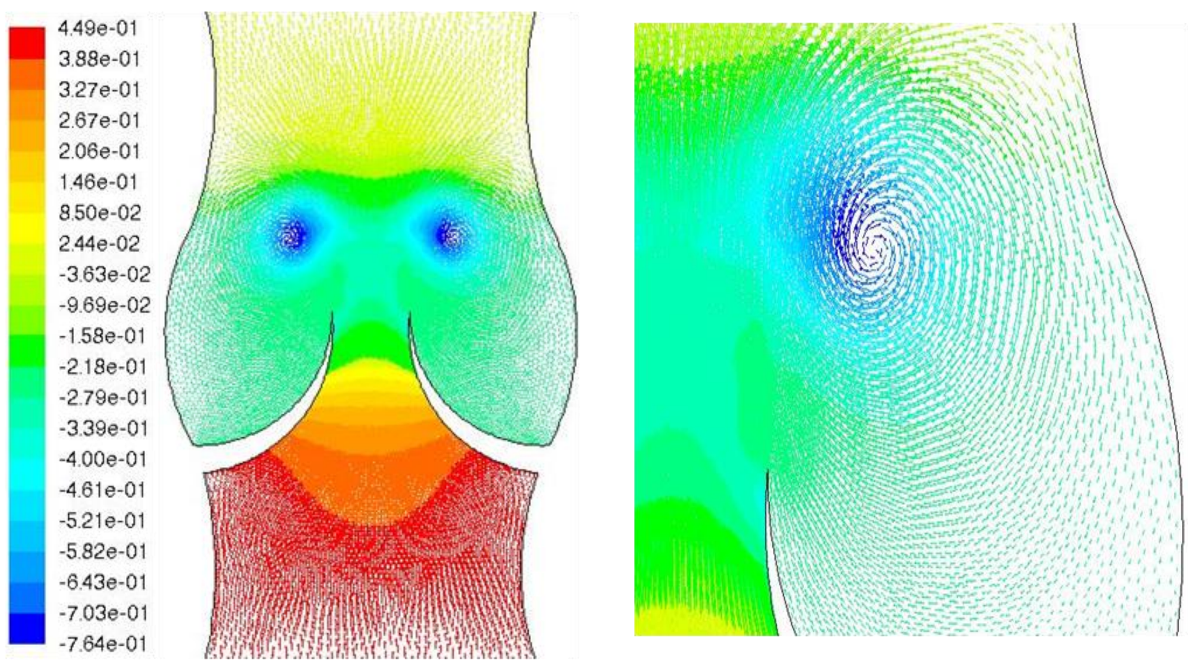


Figure 35: Velocity vectors colored with Pressure (case10)

Case11:

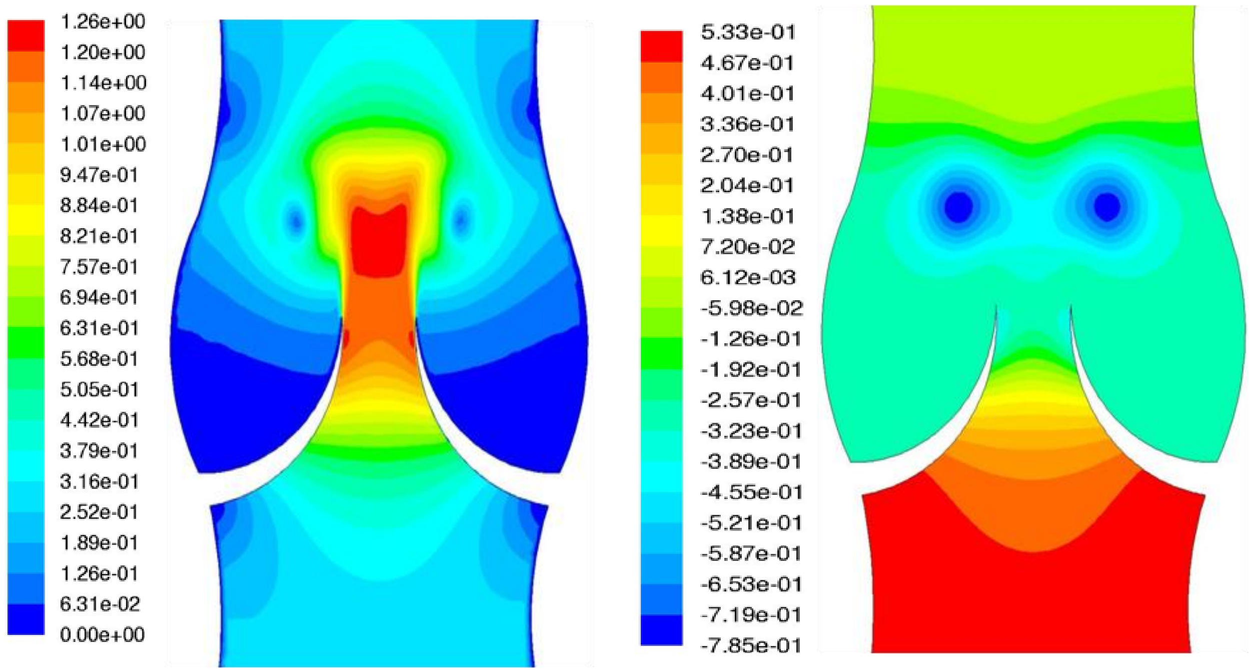



Figure 36: Velocity and Pressure Contours (case11)

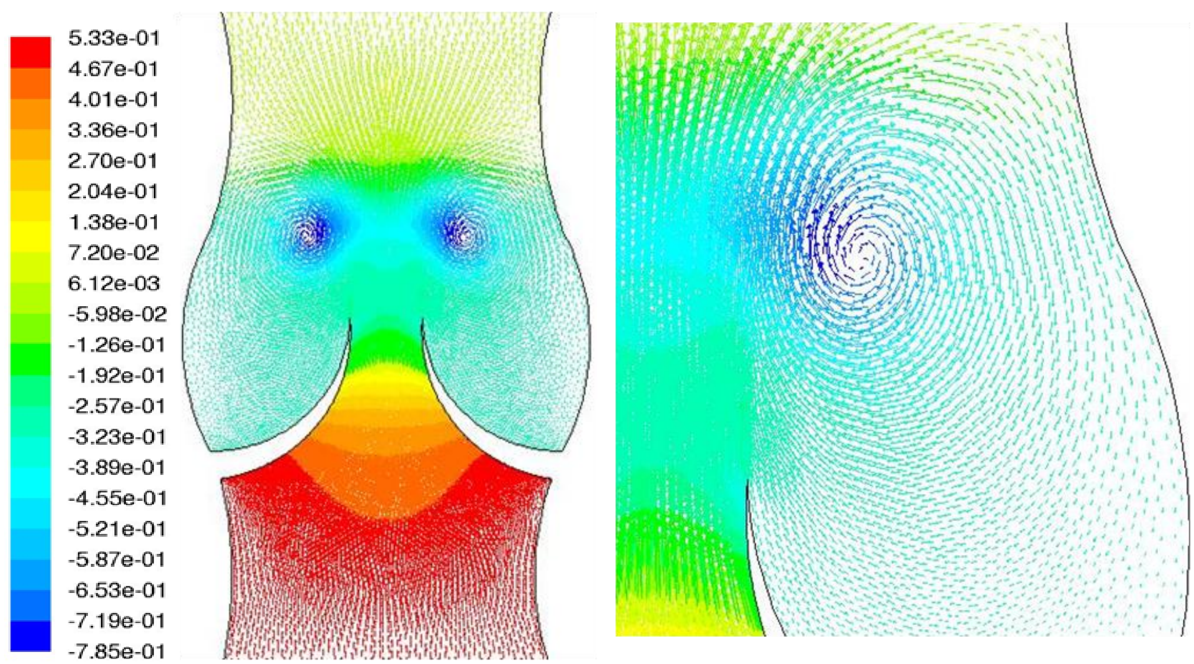



Figure 37: Velocity vectors colored with Pressure (case11)

Case12:

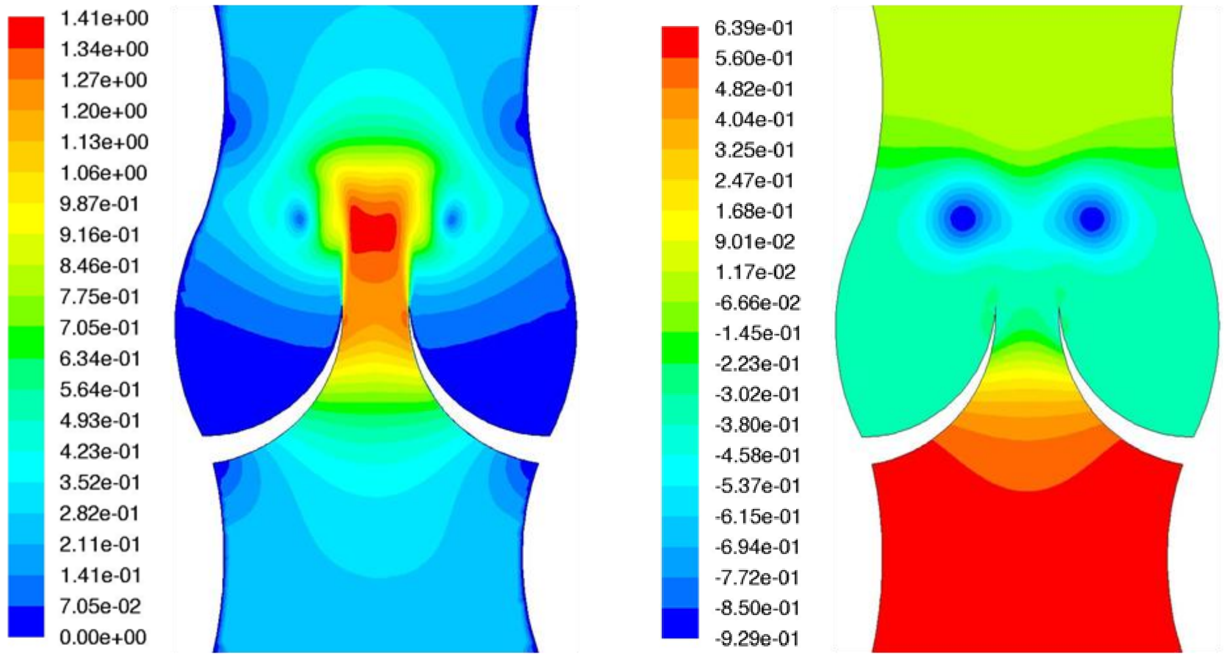



Figure 38: Velocity and Pressure Contours (case12)

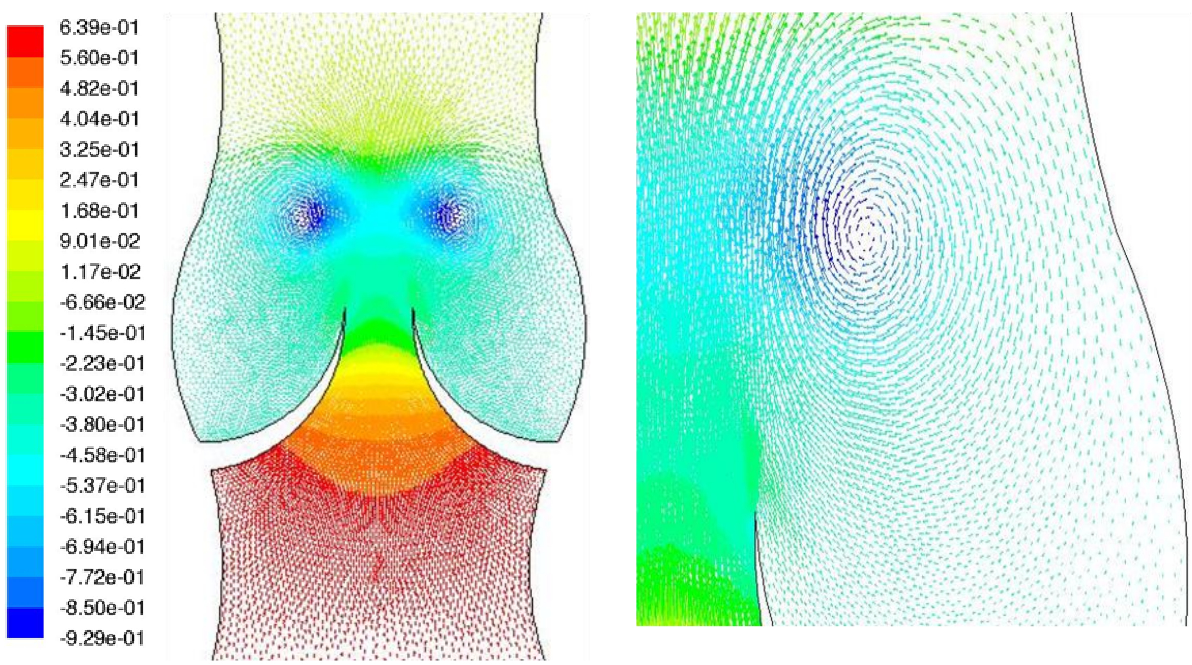



Figure 39: Velocity vectors colored with Pressure (case12)

Case13:

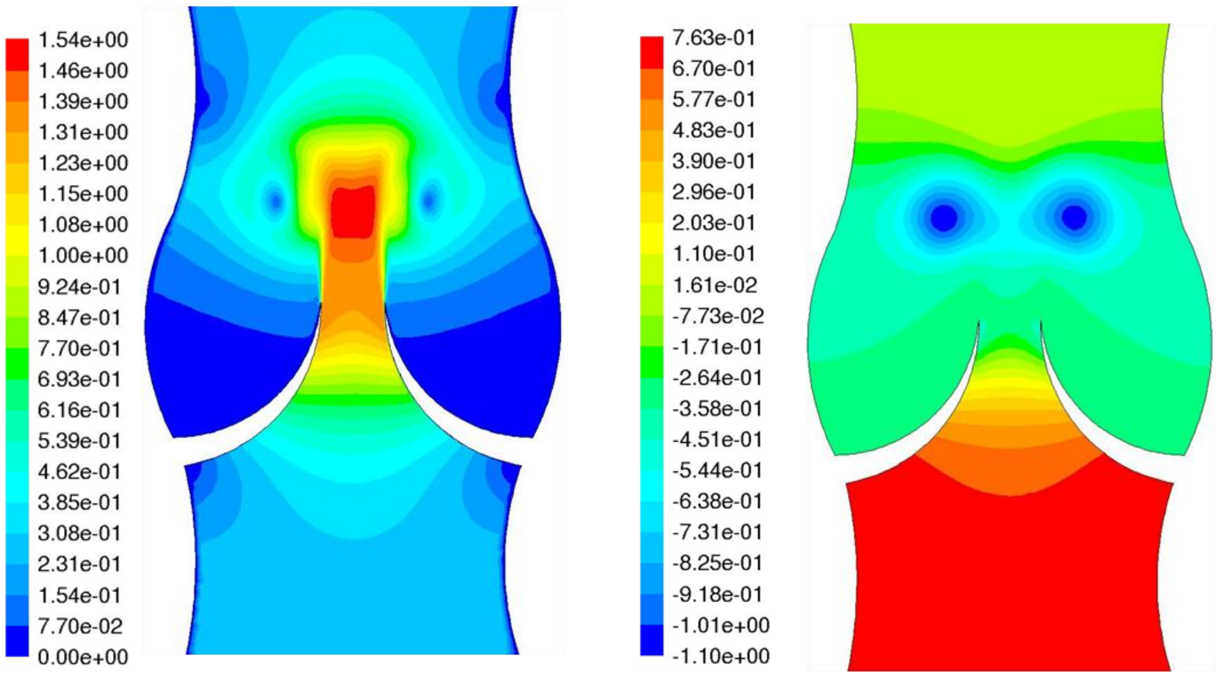


Figure 40: Velocity and Pressure Contours (case13)

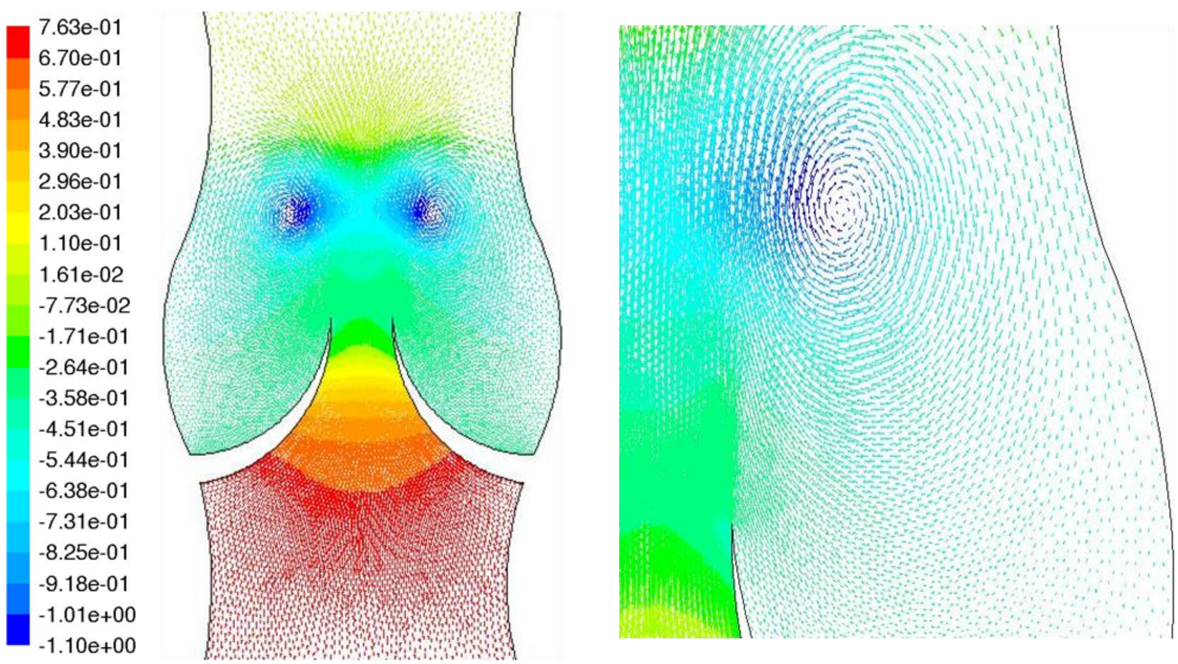


Figure 41: Velocity vectors colored with Pressure (case13)

Case14:

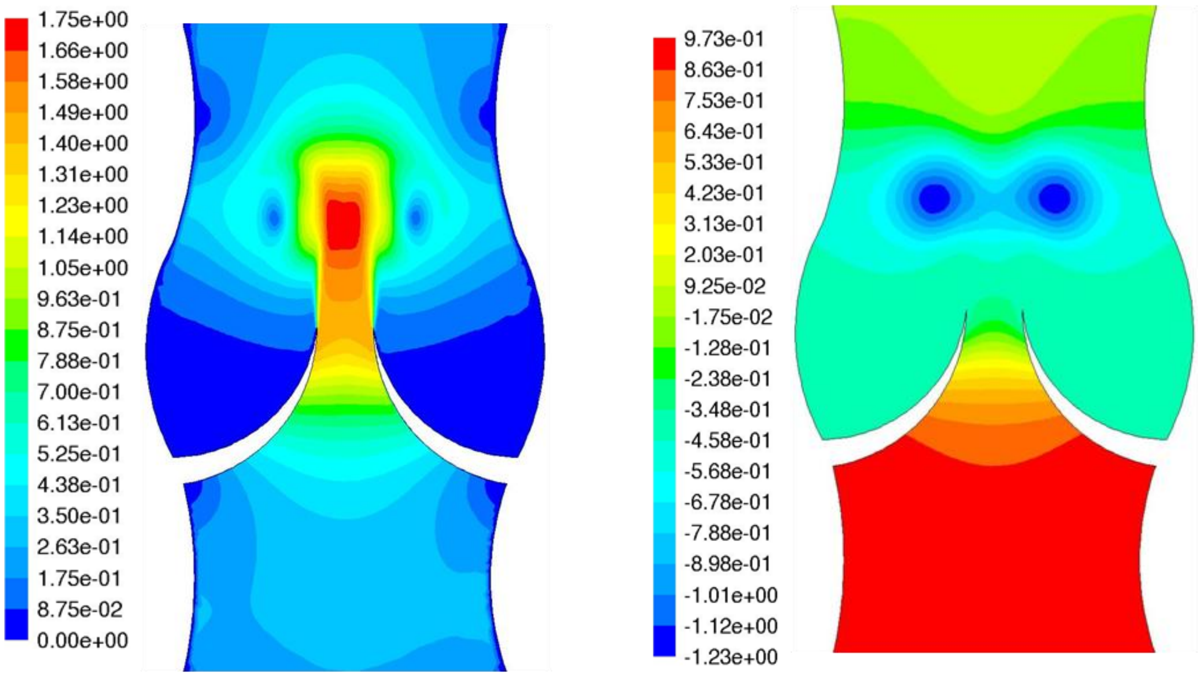



Figure 42: Velocity and Pressure Contours (case14)

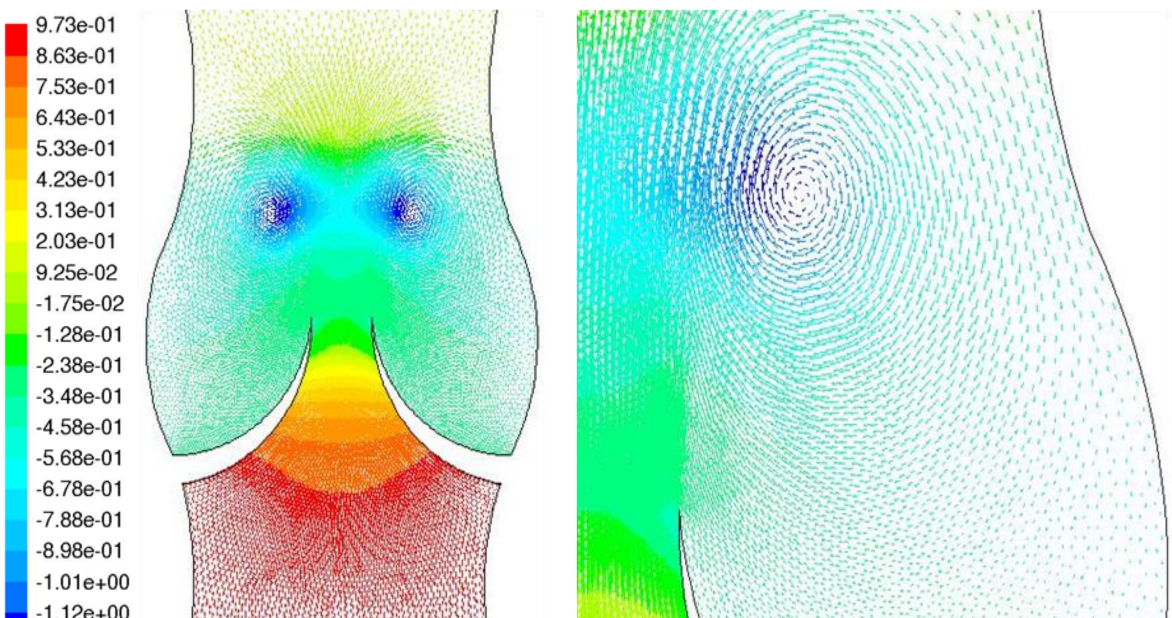



Figure 43: Velocity vectors colored with Pressure (case14)

Case15:

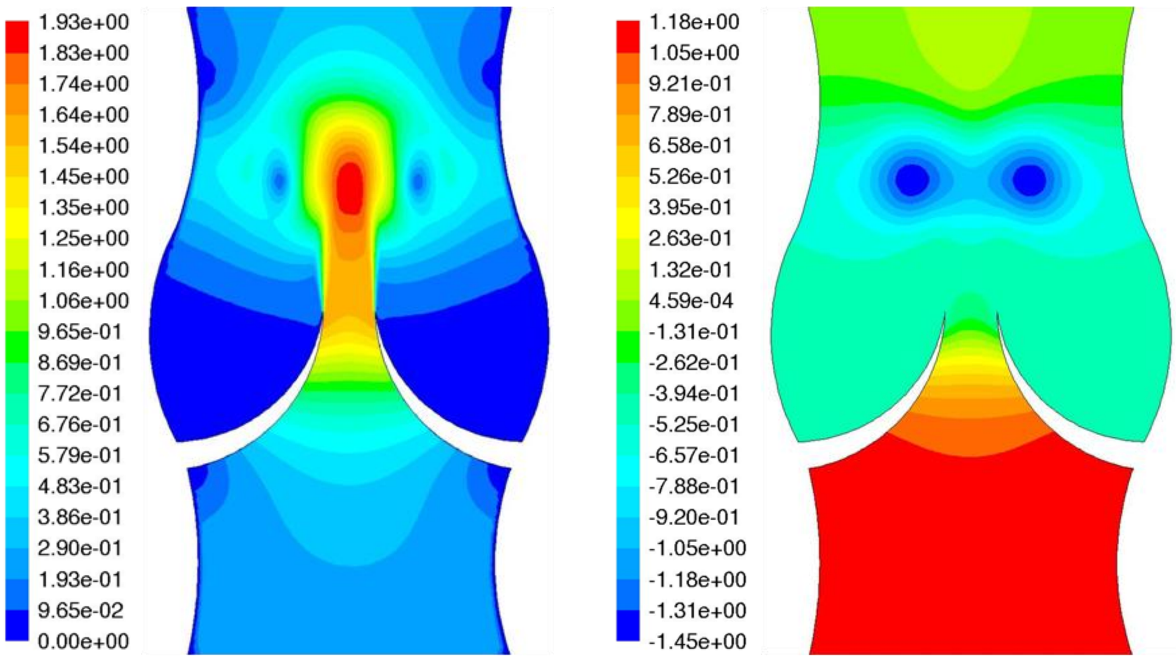



Figure 44: Velocity and Pressure Contours (case15)

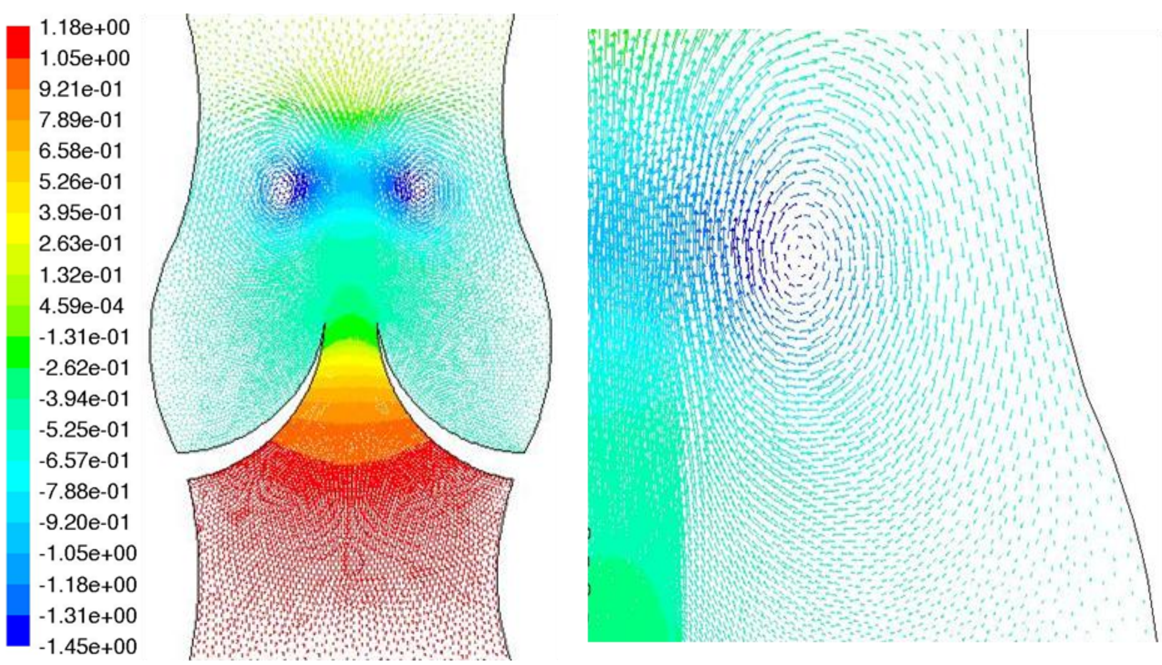



Figure 45: Velocity vectors colored with Pressure (case15)

Case16:

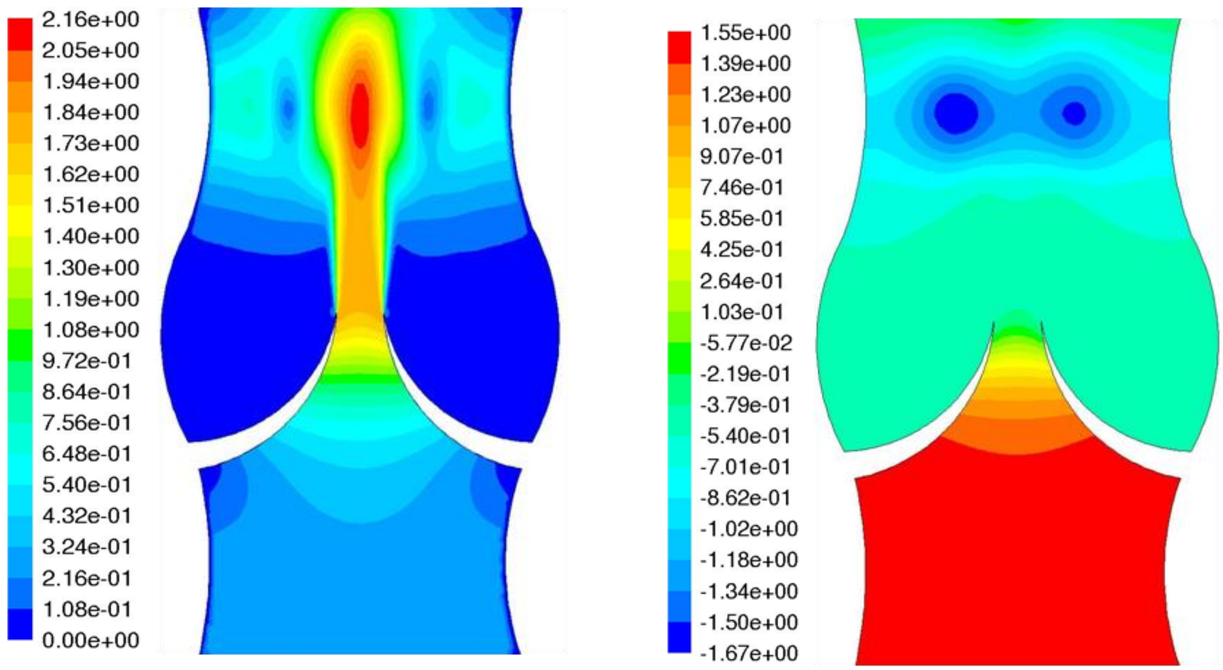


Figure 46: Velocity and Pressure Contours (case16)

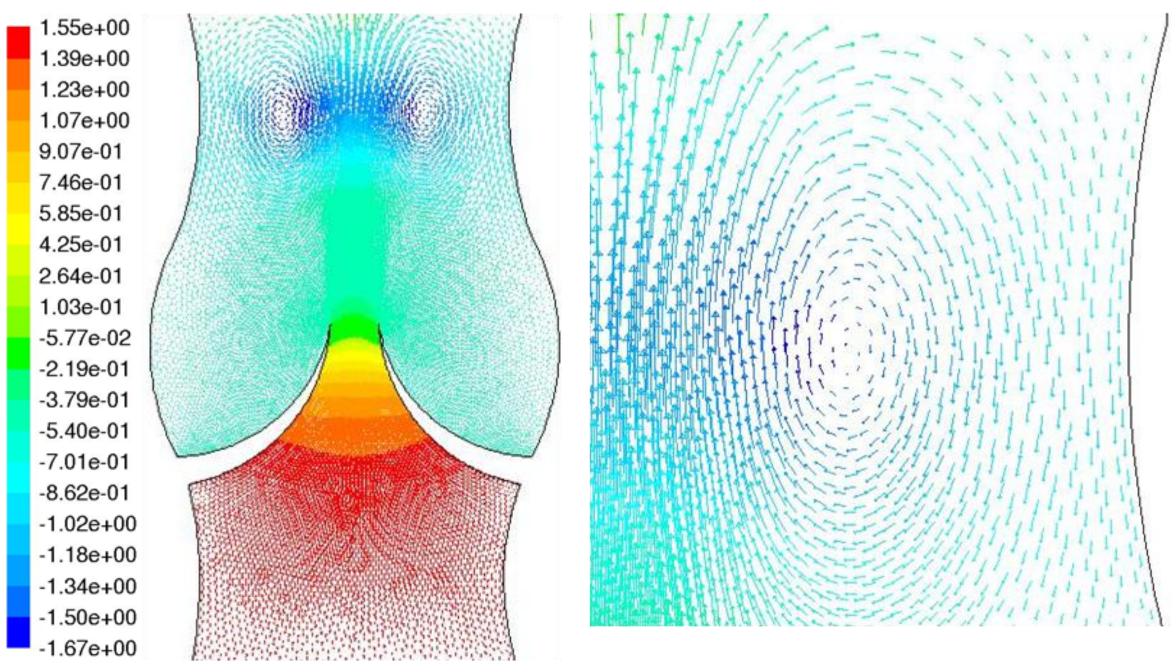


Figure 47: Velocity vectors colored with Pressure (case16)

Case17:

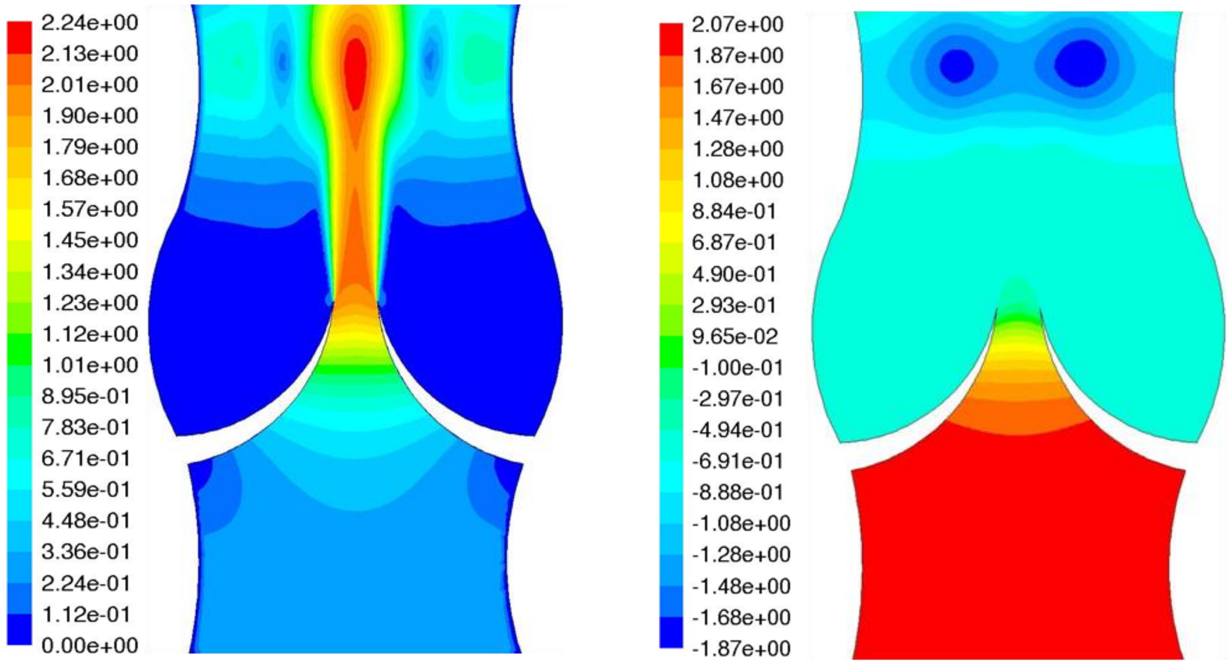



Figure 48: Velocity and Pressure Contours (case17)

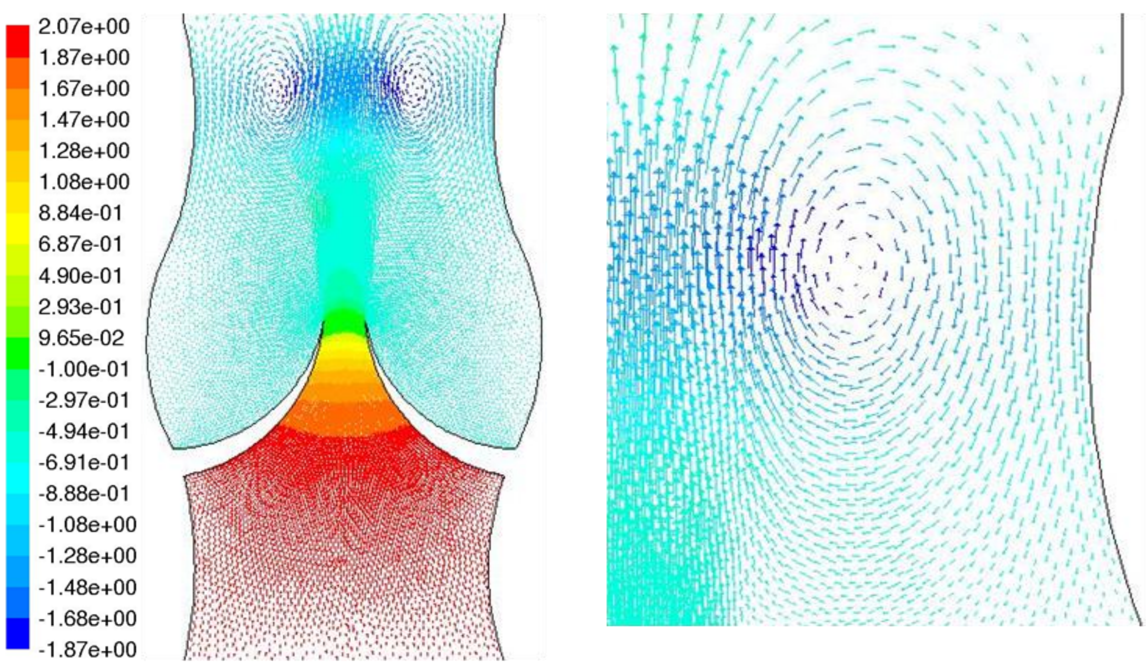



Figure 49: Velocity vectors colored with Pressure (case17)

Case18:

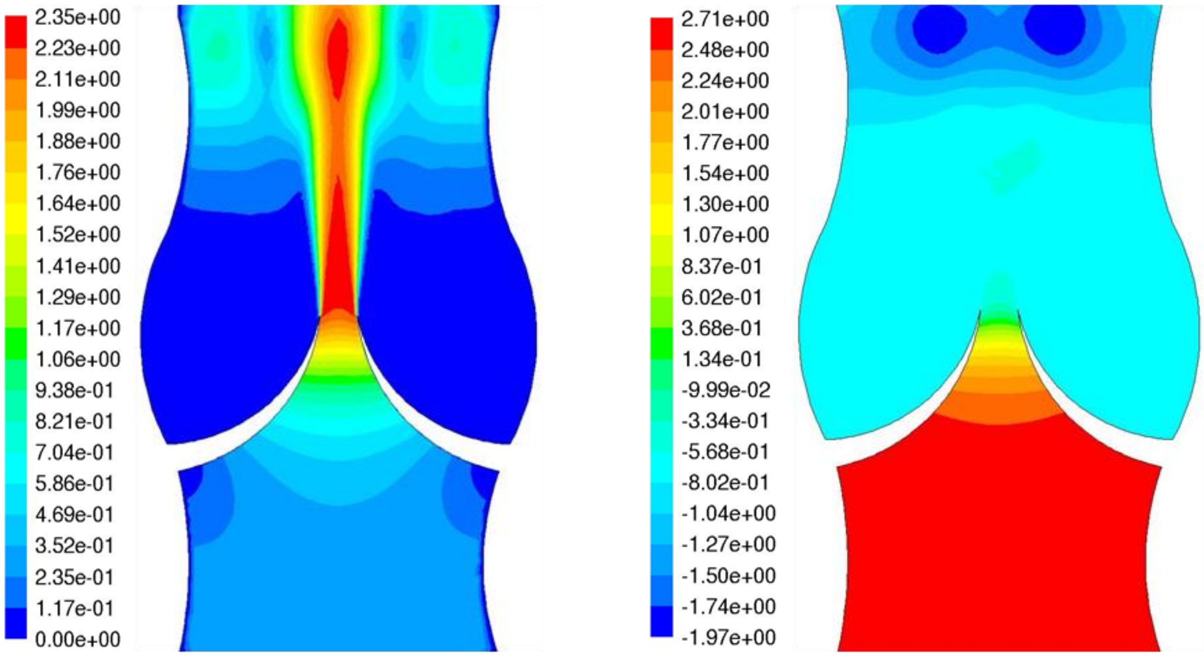



Figure 50: Velocity and Pressure Contours (case18)

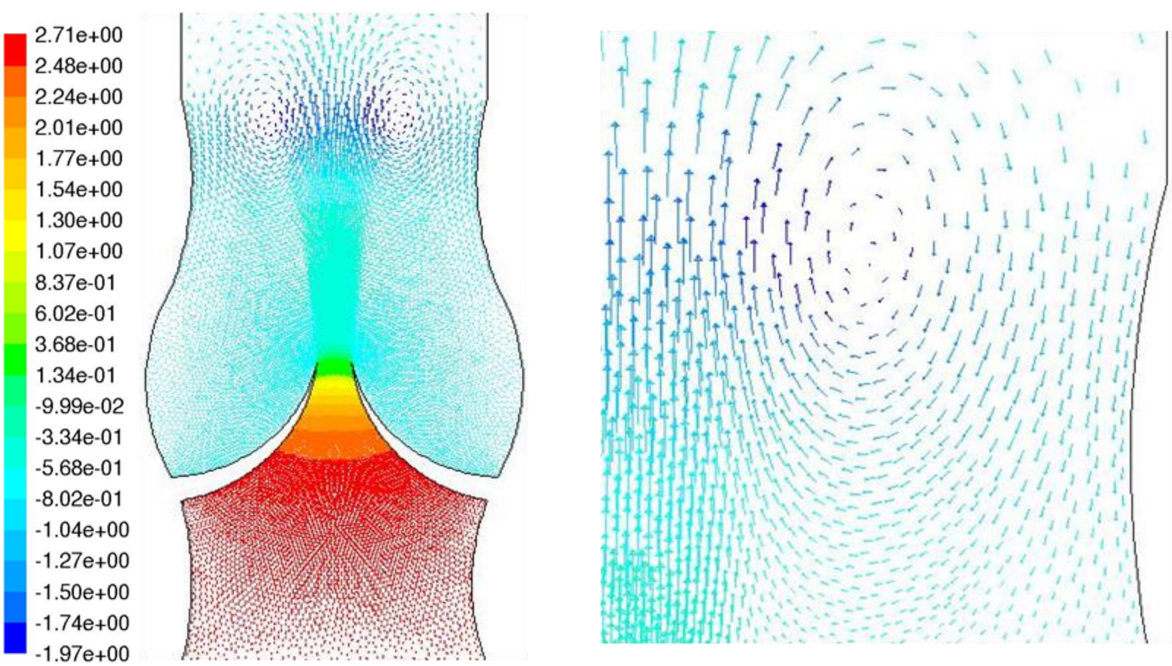



Figure 51: Velocity vectors colored with Pressure (case18)

Case19:

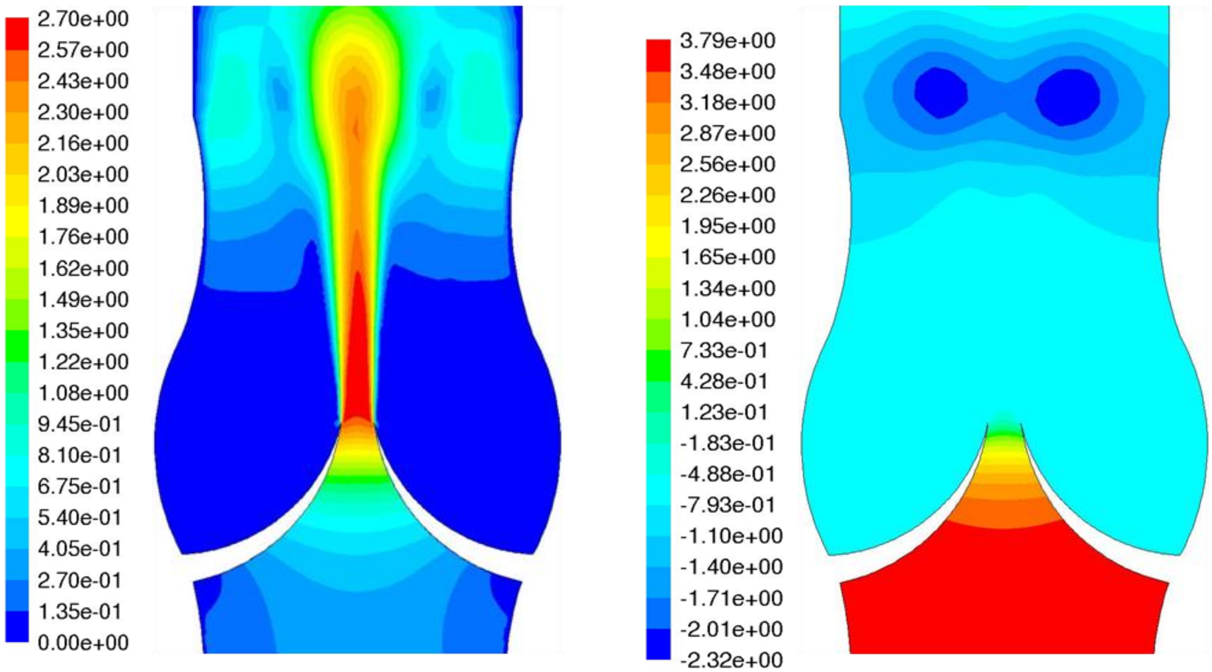



Figure 52: Velocity and Pressure Contours (case19)

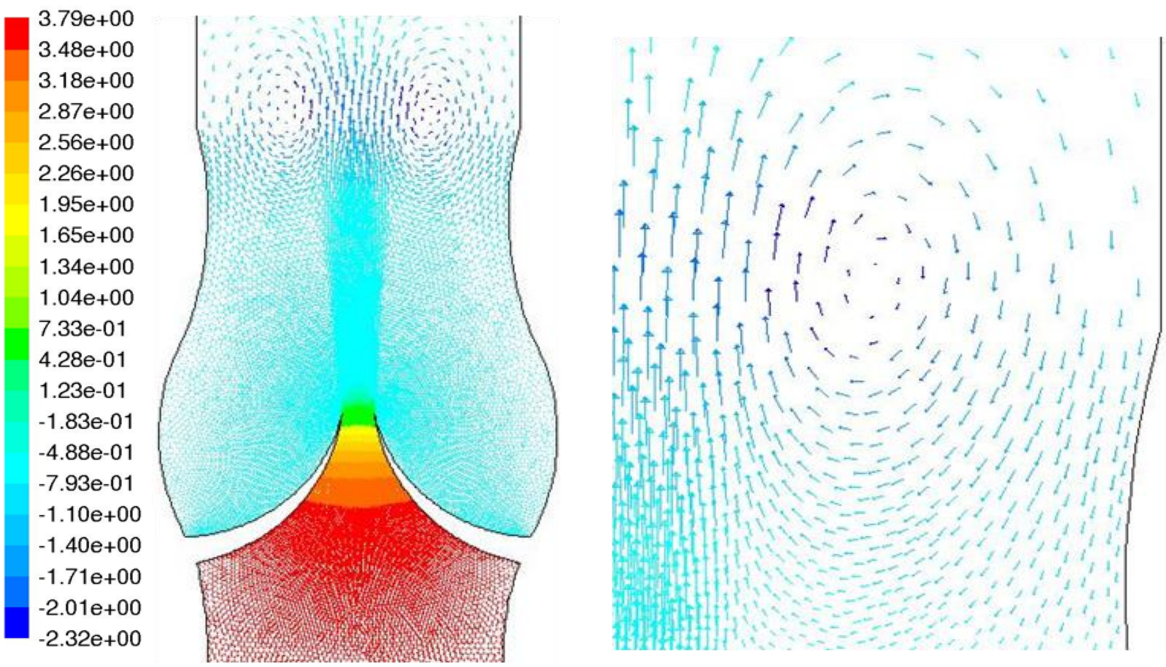



Figure 53: Velocity vectors colored with Pressure (case19)

Case20:

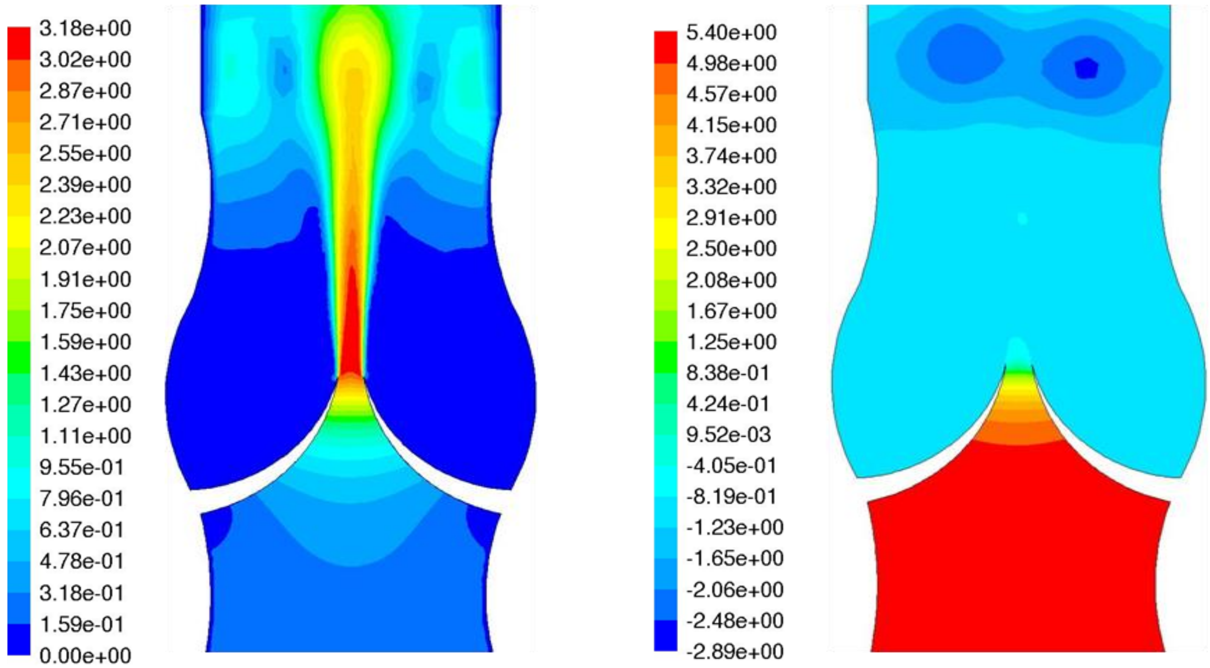


Figure 54: Velocity and Pressure Contours (case20)

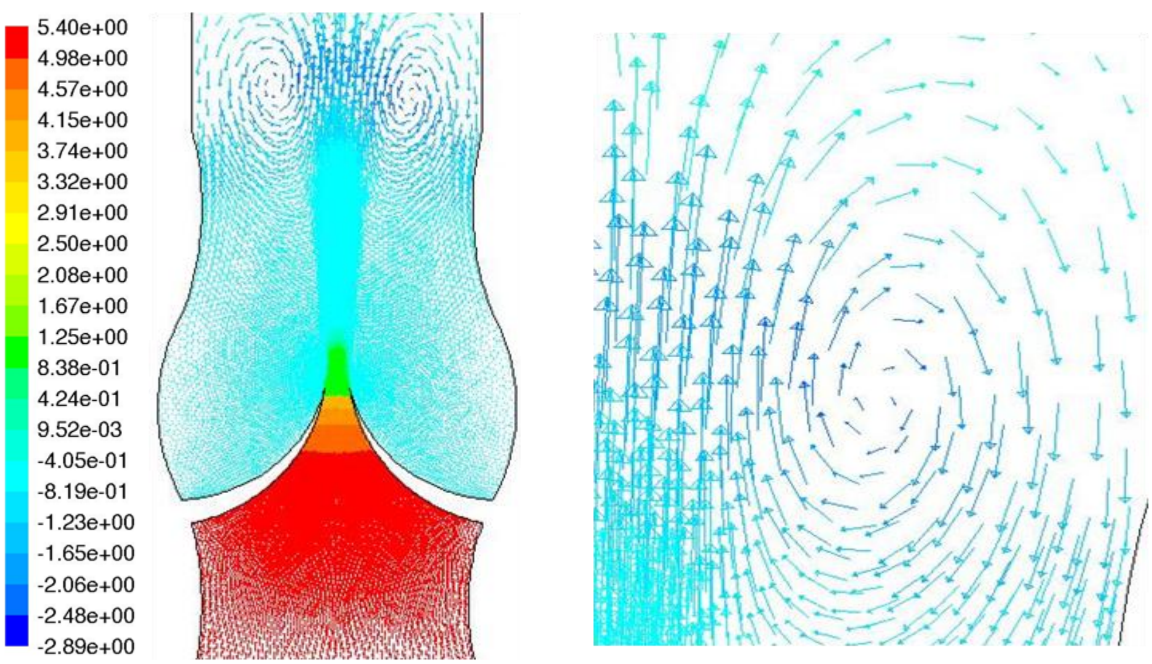


Figure 55: Velocity vectors colored with Pressure (case20)

Case21:

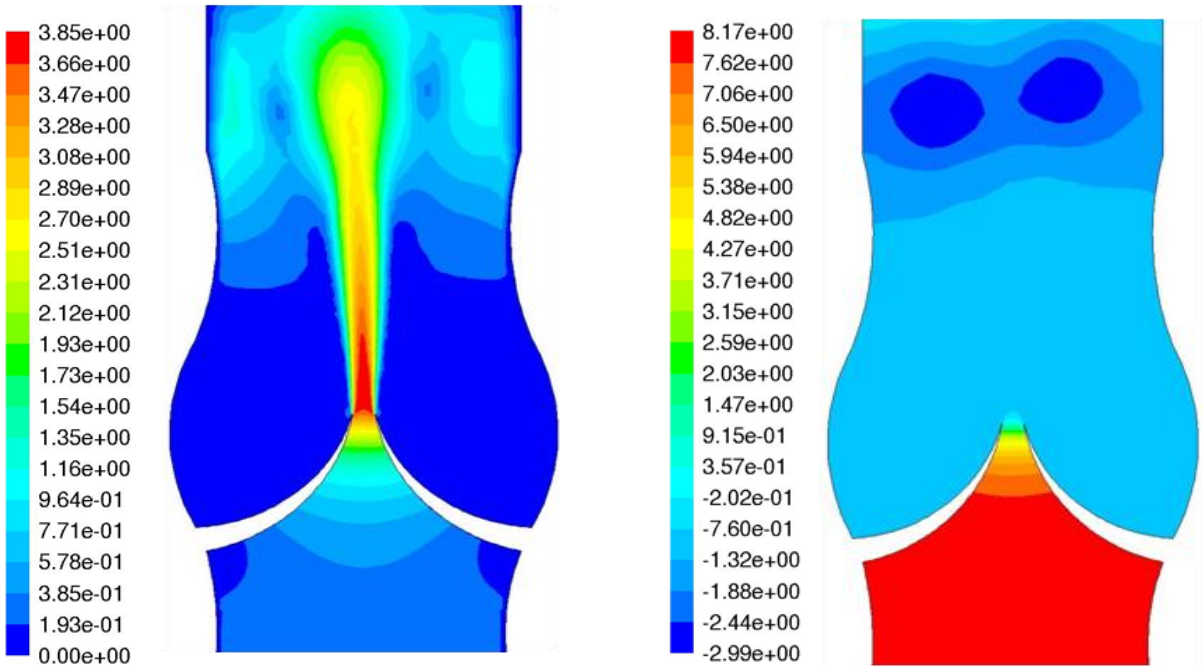



Figure 56: Velocity and Pressure Contours (case21)

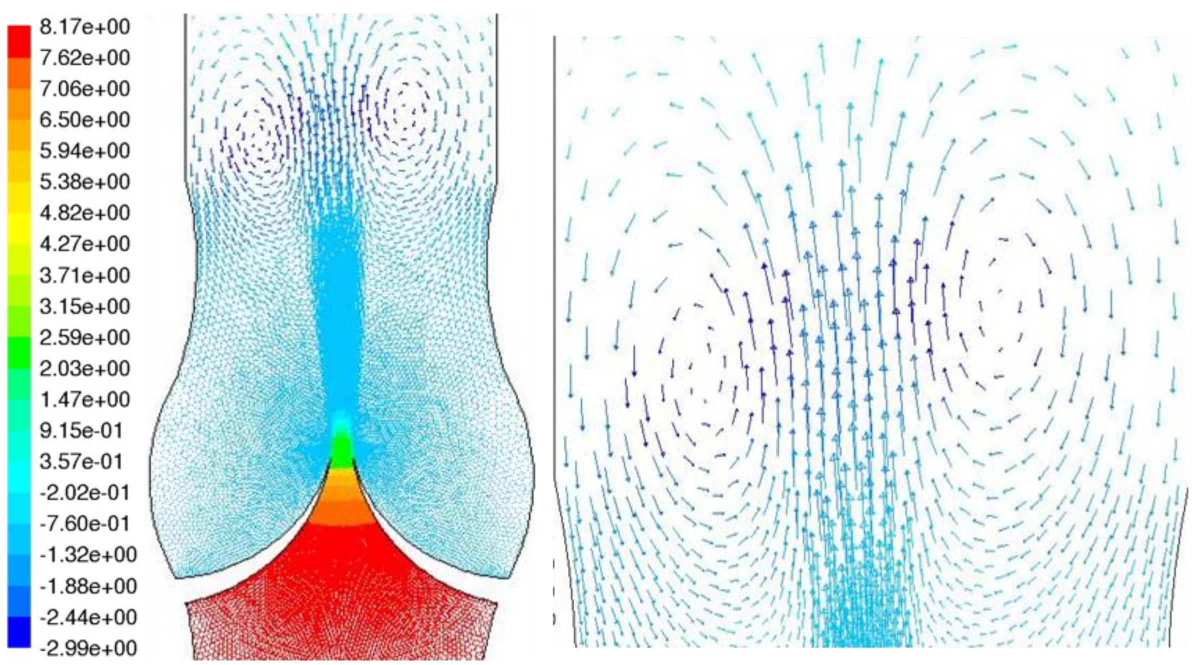



Figure 57: Velocity vectors colored with Pressure (case21)

Case22:

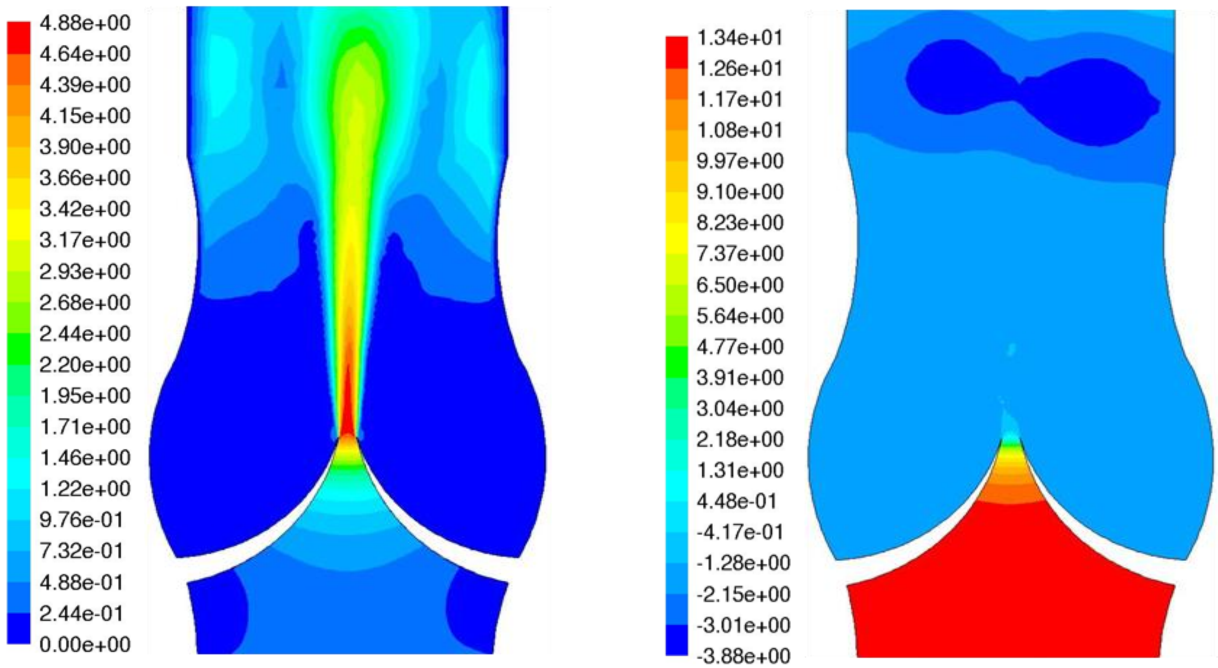



Figure 58: Velocity and Pressure Contours (case22)

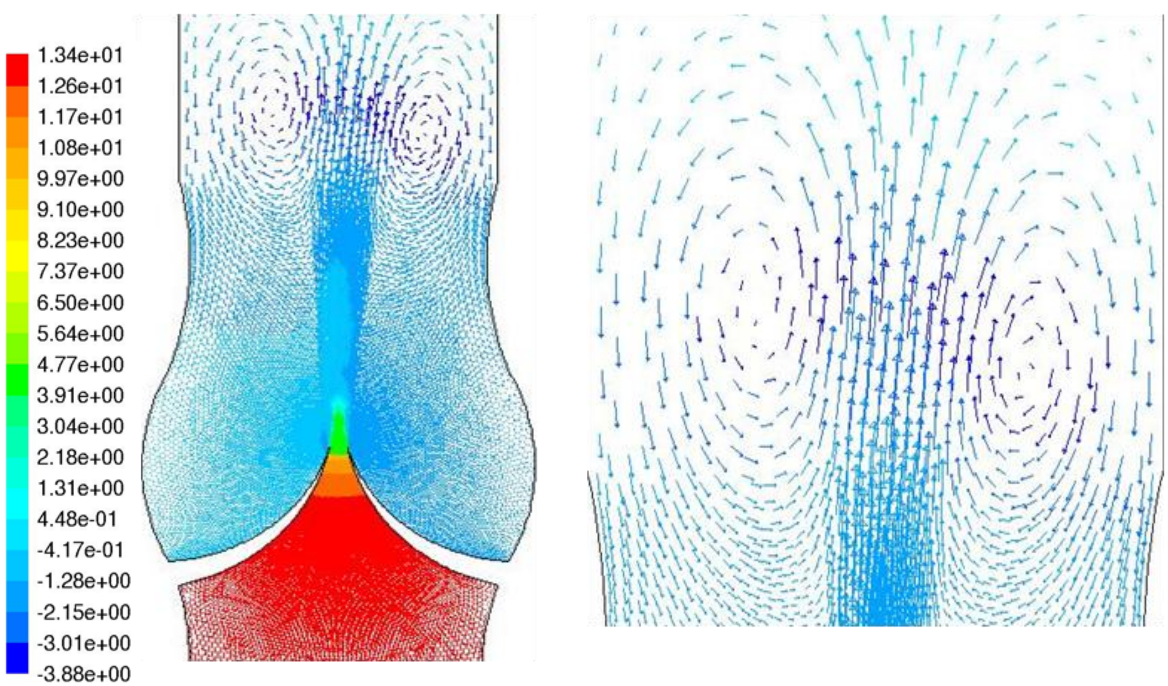



Figure 59: Velocity vectors colored with Pressure (case22)

Case23:

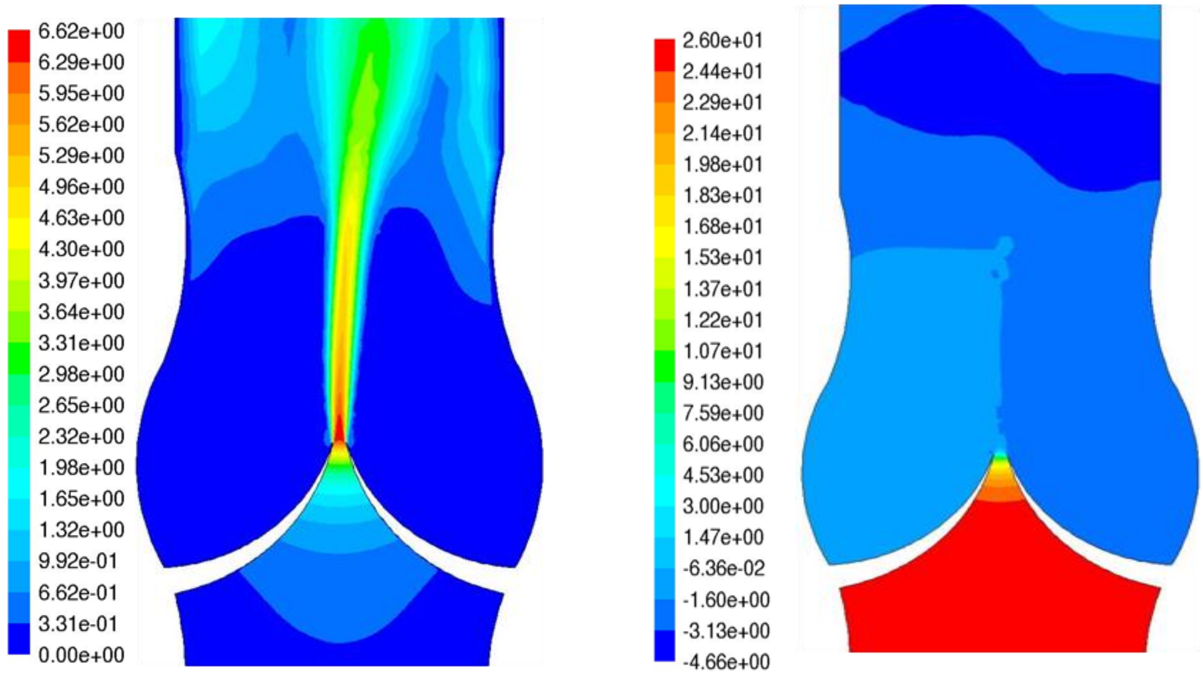



Figure 60: Velocity and Pressure Contours (case23)

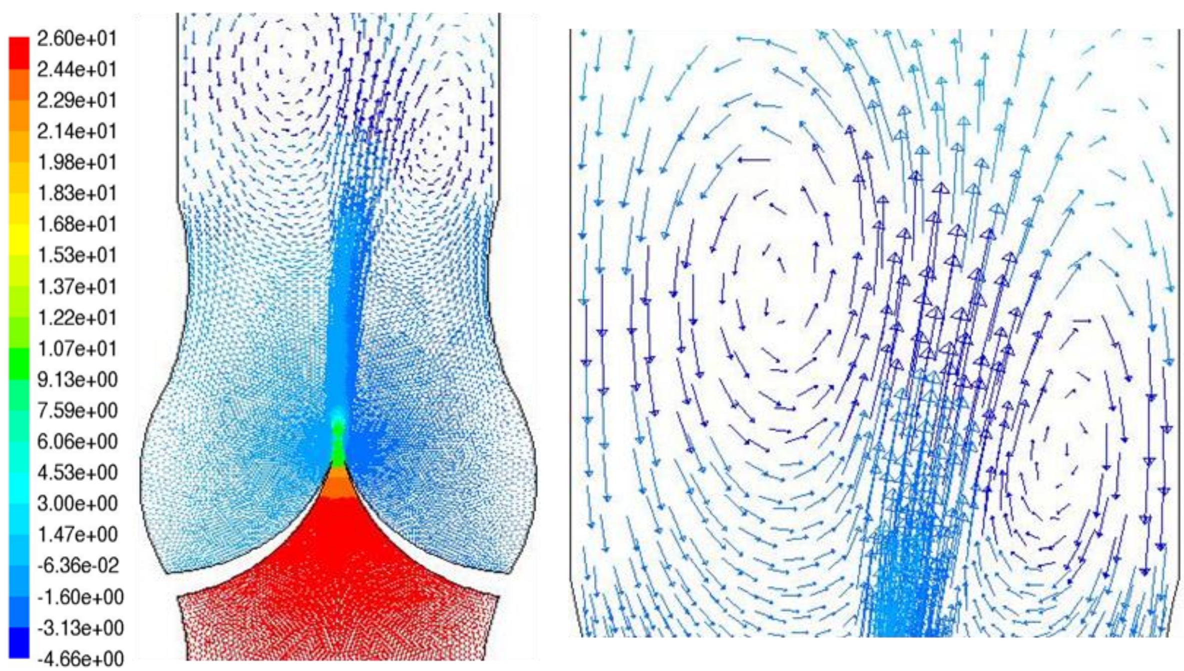



Figure 61: Velocity vectors colored with Pressure (case23)

Case24:

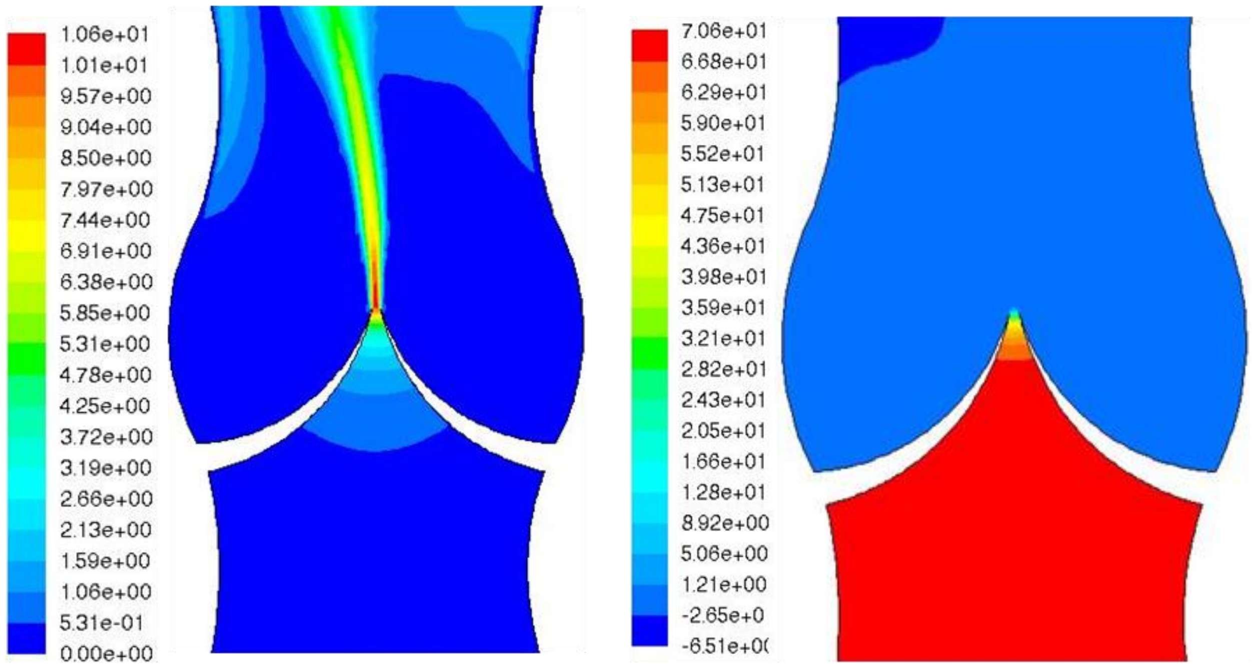


Figure 62: Velocity and Pressure Contours (case24)

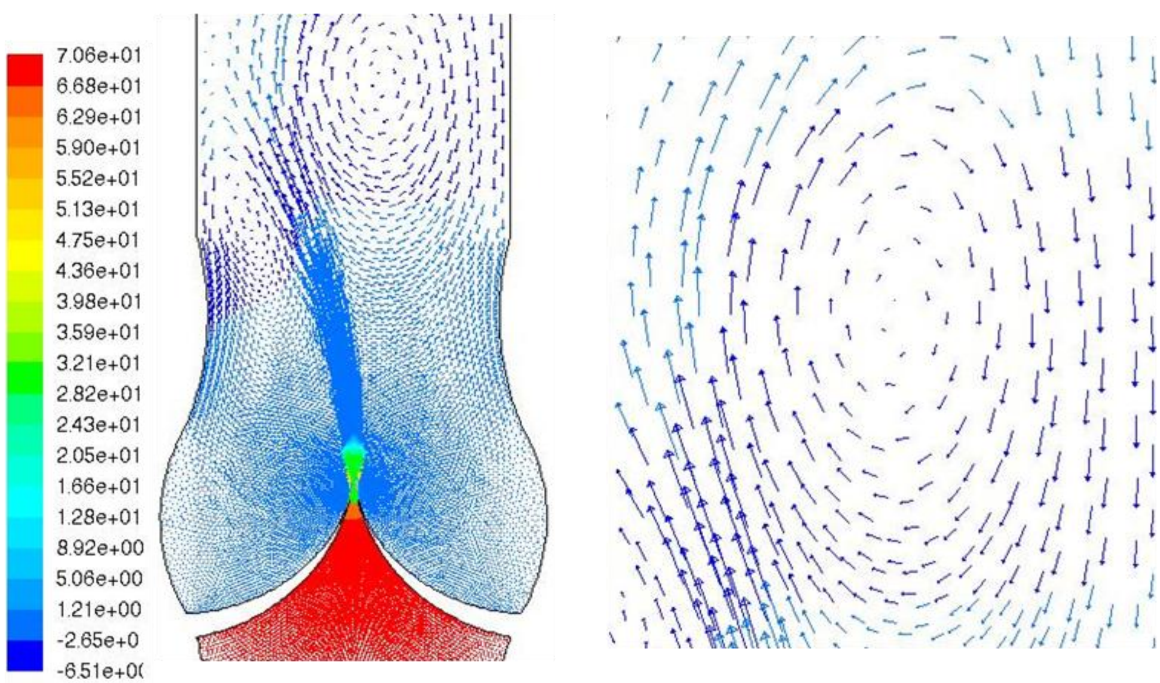


Figure 63: Velocity vectors colored with Pressure (case24)

## Curriculum Vita

Upendra Parimi was born in Hyderabad, Andhra Prasesh, India. The only son of Raghuram Parimi and Lakshmi Kumari Rajanala, he graduated from St.Patricks High School, Hyderabad, India. He then entered Guru Nanak Engineering College and affiliation of Jawaharlal Nehru Technological University in September 2004 and earned his Bachelor of Technology degree in Mechanical Engineering. He then joined the graduate school at The University of Texas at El Paso (UTEP) to pursue his masters in Mechanical Engineering in Fall 2008, as a student at UTEP he got opportunity to research in the field of Computational Fluid Dynamics. He presented a poster at the $5^{th}$ frontiers in biomedical conference held by American Society of Mechanical Engineers in September 2010.

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